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Journal of the Minnesota State Medical Association, Southern Minnesota Medical Association, Northern Minnesota Medical Association, Minnesota Academy of Medicine and Minneapolis Surgical Society

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VOL. XIII MARCH, 1930 No. 3

THE REPAIR OF FRACTURES*

CLAY RAY MURRAY, M.D.

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New York City

THIS is a subject of paramount importance to those engaged in the care of fracture cases. Some of the things I have to say will have a very familiar ring—you will find them in every textbook. Other remarks of mine, representing results of my own as yet unpublished investigations, as well as the published investigations of others working in the same or allied fields, will present a queer and interesting mixture of observed facts and attempts to explain them which seems to upset all our earlier conceptions as to the way in which bone tissue heals. Nor are we able, as a result of these newer observations, to replace our old ideas with any definite knowledge of how bone is actually formed in the healing process.

It seems, at first blush, unreasonable to present such a story to practising physicians—a destructive criticism of our present ideas, with no definite alternative to take their place. But the newer observations and the ideas which they have engendered afford us a fresh viewpoint which is of tremendous practical importance in the treatment of fractures. There is a current saying that it all depends on where you sit as to just what the picture looks like. And, as we have had to change our seats, so the picture has changed. We can begin now to understand why so many practical measures, which we have heretofore employed purely empirically, are successful, and therefore we are able to apply these measures more intelligently. We are able also to guard against, and to treat more intelligently and successfully, those conditions which we are accustomed to call delayed union and non-union. After all, it is the practical application in treatment of the ideas gained by modern research along these lines, and not the didactic discussion of them, that is important to men who are interested in the actual care of fractured bones.

All that follows deals with conditions in the adult only. The cell characteristics of the child may well be an entirely different story—there is much evidence that this is so—just as the clinical picture in children is so different from that in adults.

Let us first take up what we actually know about fracture healing. The picture of the gross in terms of visible callus or bone has not been materially improved upon since the description by Dupuytren in his Lecons Orales.[1] The whole story occupies six to twelve months, as illustrated in Figures 1 to 4.

Figure 1 represents the early provisional callus, which appears in two to four weeks. As one can see, it is similar in appearance to the plumber's wiping of a joint. It is usually fairly firm, but will slowly yield under pressure or continued stress of any considerable degree. The *x*-ray visibility is dependent on the amount of calcium present in terms of density of distribution. This may vary considerably, as may also the total amount of this callus, being least in greenstick and subperiosteal fractures and most in those with periosteum torn and with comminution of bone.

Figure 2 represents the gross picture of healing at eight to twelve weeks. The outside callus has been reinforced by a plug of callus joining the medullary canals of both fragments. There is as yet no bony union between the cortices of the two fragments. In the upper extremity this usually provides union sufficiently firm for full function without danger. In the lower extremity, because of the added function of weight bearing, it may not be sufficiently strong for full function, depending upon the weight of the indi-

*From the Department of Surgery, College of Physicians and Surgeons, Columbia University, New York. Read before the Great Northern Railway Surgeons Association, at Seattle, Washington, July 5, 1929.

vidual, the calcium density of the callus, the location of the fracture from the viewpoint of weight-bearing mechanics, etc.

Figure 3 shows the condition present at the end of four to six months. The outside callus has diminished

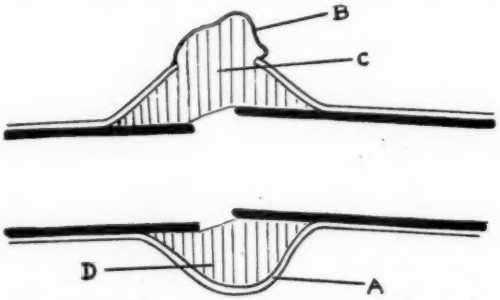

Fig. 1. *A.* Stripped-up periosteum. *B.* Torn periosteum. *C.* Callus. *D.* Callus.

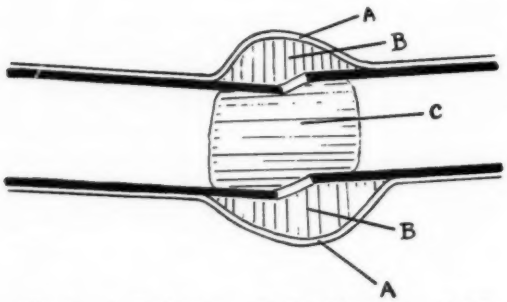

Fig. 2. *A.* Periosteum. *B.* Outside callus. *C.* Medullary callus.

in size and increased in density; the medullary canal shows a bony plug with visible vascular channels, and the cortices of the two fragments are joined by a bony callus. This is so-called solid bony union. Subsequent changes deal only with the architecture of the bone. That architecture is supposed to be in accordance with the stress and strain to which the bone is subjected—the so-called Wolff's law. As Carey[2] has shown, the factors involved may be many and varied. If subjected to the stress and strain normal for the bone involved, the normal architecture will be regained. If not, an architecture will be attained which is in accordance with the modified stress and strain placed upon the bone. This is not of necessity a mysterious or complicated process. A steel girder or column subjected to a given stress and strain will arrange its lines of crystallization in accordance therewith, and if the stress and strain be changed for a sufficiently long time it will rearray those lines to meet the new conditions. It is to bring about this resumption of normal architecture that fractured necks of the femur, after healing has taken place, are put on partial weight bearing, protected against compression of the callus and resultant angulation and coxa vara deformity by a walking caliper for some months. We know from experience that the homogeneous callus at this crucial point as regards weight-bearing is frequently unable to bear the burden until the lines of force transmission in the neck of the femur are at least partially restored by the action of Wolff's law.

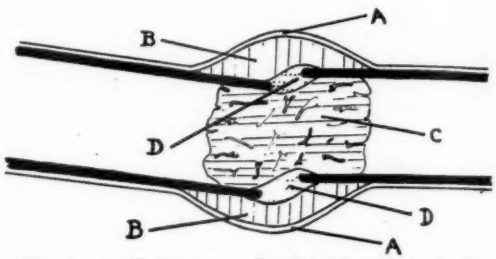

Fig. 3. *A.* Periosteum. *B.* Outside callus. *C.* Medullary callus. *D.* Cortical callus.

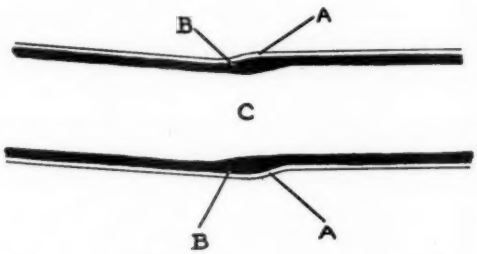

Fig. 4. *A.* Periosteum. *B.* Cortex. *C.* Medulla.

Figure 4 represents the final result nine to twelve months after fracture. The bone is healed and has regained its normal architecture, completing the picture in the gross.

Let us now turn our attention to the study by the microscope of what actually occurs in bone healing. On this score the newer observations have revealed facts which can be demonstrated in the illustrations which follow. These facts have changed completely our conception of how bony injury heals, and have done much to undermine our ideas as to how bone replaces the mere healing to give solid bony union.

The illustrations comprising Figures 5 to 22 are taken partly from articles published by Bancroft*[3,4,5,6], partly from his own private collection and partly from the files of the Department of Surgery, Columbia University. The observations recorded and the conclusions drawn are partly Bancroft's own as published, and partly mine as made in a further study of his work in conjunction with my own experimental study of fracture repair as cited below.[7] (The originals of the illustra-

*I am indebted to Dr. Bancroft for his courtesy in allowing the use here of his illustration. It is a pleasure to acknowledge my indebtedness to him not only for the illustrations and his own observations, but for the stimulus derived from his work to further study of the question.

tions were previously published in the following periodicals: No. 18, Am. Jour. Med. Sci.; Nos. 7, 9, 19, Arch. Surg.; No. 8, Ann. Surg. and New York State Jour.)

Figure 5 is diagrammatic of the details of the gross

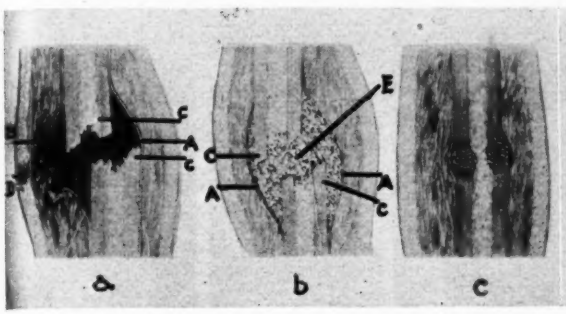

Fig. 5. *A.* Periosteum, real or apparent. *B.* Torn and infiltrated muscle. *C.* Broken bone ends. *D.* Infiltrated subcutaneous tissue. *E.* Callus.

shown in Figures 1 to 4. "a" represents the pathology at the time of fracture. It shows the broken ends of the bone (C), which we shall show later undergo death and autolysis to a variable degree. The periosteum is represented as intact on one side (A) and torn on the other. It is obvious that all granulation tissue on the sound side will of necessity be derived from the only soft parts available, which are the under surface of the periosteum, the so-called endosteum of the broken edges of the bone and the marrow tissue of the fragments. On the torn side it is equally obvious that the torn and lacerated muscle and fascial planes are allowed access to the fracture site, and granulation tissue produced in them can grow in to the site of the fracture. The extent and location of the hemorrhage and exudation at the fracture site is also worthy of note because of its distribution and the fact that the swelling produced causes a variable amount of circulatory and lymphatic disturbance (B) (D). These factors will all be dealt with later as to their influence on the course of the healing process. "b" represents the callus healing stage. The periosteum will be noted as apparently restored where it was torn before. As we shall show later, with tearing as extensive as here shown, the restoration of periosteal integrity is apparent and not real, the bridging tissue being derived from connective tissues outside the periosteum, viz., muscle and fascial planes. The only morphological difference observable is the absence of the very vascular inner layer present in normal periosteum. Despite its source and this morphological difference, the new tissue functions effectively, and, so far as we can tell, in a normal manner as periosteum. This detail will be referred to again in its bearing on the question of the specificity of the periosteum as a tissue. The bony callus represented in this diagram shows numerous vascular channels, which we will show later to be at right angles to the long axis of the bone and to be the vessels of granulation tissue derived from outside the bone, in part at least. The question of the origin of the calcium deposited in that tissue to form bone will also be a matter for clarification. "c" represents the healed state with restoration of the bone architecture.

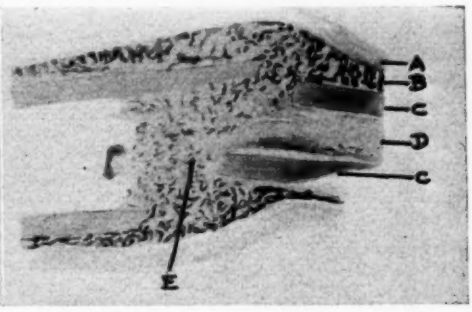

Fig. 6. *A.* Periosteum. *B.* Subperiosteal bone. *C.* Cortex. *D.* Medulla. *E.* New bone, with Haversian system at right angles to shaft.

Figure 6 is diagrammatic of a late stage of callus repair and is a little more detailed representation of "b" in the preceding figure. Its captions explain it and it holds the same points of interest as were detailed in Figure 5 "b."

Figure 7 is a microphotograph of the healing of a comminuted fracture. In it can be noted the numerous fragments with microscopical evidence of death, as shown by lack of nuclear staining, and death of the ends of the cortical bone on either side of the region of comminution, with new bone production taking place about these areas of bone death. The direction of the vessels in many of these areas agrees very accurately with the diagrammatic representation in Figure 6, and the point brings up similar questions as to the source of the tissue of origin and of the calcium involved. The formation of the pseudoperiosteum and the absence of the deep vascular layer in it is also shown. The fact that the new bone formation occurs primarily about areas of microscopically dead bone is also a fact to be kept in mind.

Figure 8 represents a late stage of experimental comminuted fracture. The experimental technic was that of Bancroft in the study of repair of bone defects by the use of chip grafts. About one inch of a dog's radius was excised with its periosteum. The ulna was left intact to maintain the gap. The excised portion was scraped free of periosteum and fragmented and the small fragments were freed from so-called endosteum in so far as possible by scraping and then dropped back into the gap and the muscles sutured lightly to hold them in place. While devised for the study of chip transplants, the procedure reproduced realistically the condition of comminuted fracture with the fragments separated from the periosteum and with wide tearing of the latter. Studying the microphotograph from the viewpoint of a comminuted fracture of this type of 141 days duration, we see at A the broken

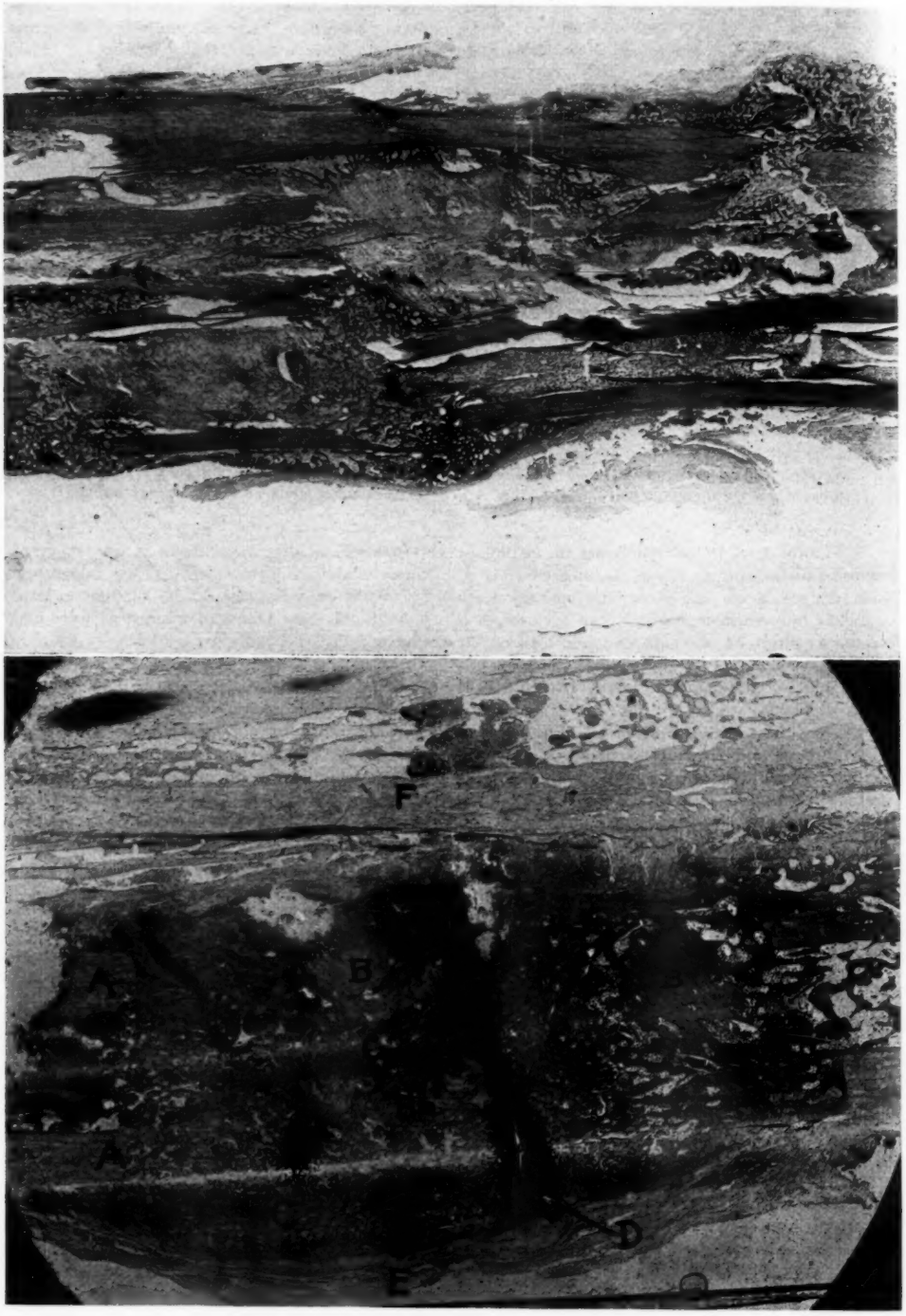

Fig. 7 (*above*). Healing comminuted fracture.

Fig. 8 (*below*). *A.* Broken bone ends (radius). *B.* Fragments. *C.* New Bone. *D.* Cartilage. *E.* Pseudoperiosteum. *F.* Ulna (intact)

bone ends with the comminuted fragments B lying between them, both showing microscopic evidence of death. The whole is amalgamated by a variegated structure comprising new bone, cartilage and connective tissue extending laterally on both sides beyond the bone limits for a considerable distance. We see that the new bone formation is to be observed principally about the bone fragments and the broken ends of the bone. We note that for a short distance from each broken bone end the direction of the vessels is in the long axis of the bone, but that in the intervening area, comprising four-fifths of the original gap in the bone, the vessels are at right angles to the long axis of the bone. In earlier sections these vessels are shown to be continuous with and apparently a part of the granulation tissue proceeding from the soft parts. These facts are of significance in regard to the question of the tissue of origin of the healing process, the source of the calcium utilized to transform that tissue into bone and the rationale of that transformation. We see also the formation of a pseudoperiosteum by the surrounding soft parts as in previous figures. We note also the diverse types of tissue present—cartilage, connective tissue and new bone—a matter into which we must inquire more deeply as we go on.

Figure 9 represents a low power microscopic picture (x 150) of one of the included fragments shown in Figure 8. The areas of obviously dead bone are denoted by the absence of nuclear staining, although the cell spaces are plainly apparent. One notes also that the vessels are apparently viable, as shown by normal nuclear staining of the vessel wall. Are these vessels the original ones of the bone fragment "revivified" by a new nutritional supply, or are they new vessels? Study of similar sections from a few days onward to the stage here shown makes it apparent that these vessels are new; that they are the vascular buds of granulation tissue invading the chips of hard bone by the only available channels—the pathways of the old vessels—just as they invade the fibrin network at the site of fracture. These vascular buds consist of the capillaries surrounded by a thin layer of accompanying fibroblastic cells. In the new channels which they occupy, one notes that live "bone cells" seem to exist in three or four layers in the cell spaces in the immediate region of the vessel and nowhere else. Are these "revivified" bone cells brought to life by the reëstablishment of circulation? If the site of this picture were out in the healing soft parts we should state immediately that they were the fibroblastic cells surrounding the capillaries to make so-called granulation tissue. And that is all they are here, invading in their progressive growth the only available space—the empty cell spaces in immediate proximity to the vessels which they accompany. And they are bone cells only as regards the fact that they occupy the cell spaces in the bone. Morphologically and by staining reaction they are not to be distinguished from fibroblasts.

Figure 10 is a higher power (x 375) of a small section of the picture shown in Figure 9, and brings out all the details more clearly. The morphological appearance of the cells, their distribution and the presence of empty cell spaces in the avascular areas are readily noted in this microphotograph.

Figure 11 represents a section taken through the site of a linear fracture in a dog which was given immediate treatment. The section was made after two weeks, at which time gross healing was apparently accomplished. At (B) will be noted the end of one bone fragment with absence of nuclear staining and empty cell spaces. This is "dead bone," comparable to the state of the bone chips in previous illustrations. At (E) there is to be seen "cyst" formation in the bone fragment end—an evidence of bone atrophy or the demineralization process. This is important. Some bone is killed by the trauma and by the vascular disturbance attendant upon it, and that dead bone is demineralized. This is comparable to the autolysis of any dead tissue through the action of ferments freed or activated by tissue death. What is important is the question as to what happens to the calcium freed by the demineralization of the bone and as to the form in which it exists. Circulation in this injured area is sluggish and slow, due to mechanical as well as inflammatory factors. This must be kept in mind. We do not know the form in which calcium exists in the bone or in the blood or tissue fluids. At (D) will be observed normal bone. (C) designates the callus lying between the bone ends. The apparent direction of growth is not from bone end to bone end, but is across the gap, perpendicular to the long axis of the bone fragments. This, as we have noted, is the direction of growth of the granulation tissue which later becomes bone in the experimental comminuted fractures cited above. (A) represents an area of cartilage present in the callus, although no pre-existing cartilage can be premised, except on the basis of cell rests of embryonal origin. In this cartilage area the cell types range from frank cartilage cells to cells indistinguishable from the fibroblastic type, and the ground substance varies from the typical blue staining hyaline substance to that which is indistinguishable from the adjacent collagen fibers of ordinary connective tissue.

Figure 12 is a microphotograph of a three-day human callus recovered at operation. It is of extreme interest in that it shows typical new bone formation at this early stage in new connective tissue. In the upper right hand and lower left hand corners we can see fairly dense areolar tissue. The center of the picture is occupied by a loose meshed tissue in which calcium has been deposited to form a lace-like structure of new bone. The cells lying in this mesh of calcium are identical morphologically with the fibroblasts lying in the tissue outside. The calcium matrix merges into the basement substance of the areolar tissue at points so imperceptibly that it is extremely difficult to tell just where calcium deposition begins. In the lower right hand portion the typical appearance of new forming bone is present with linked, roughly ring-like areas of calcium surrounding small areas of areolar tissue; in the latter the capillaries are present. This will be shown in detail in later illustrations.

Figure 13 is a drawing made from a microphotograph of a nine-day human callus which shows more clearly

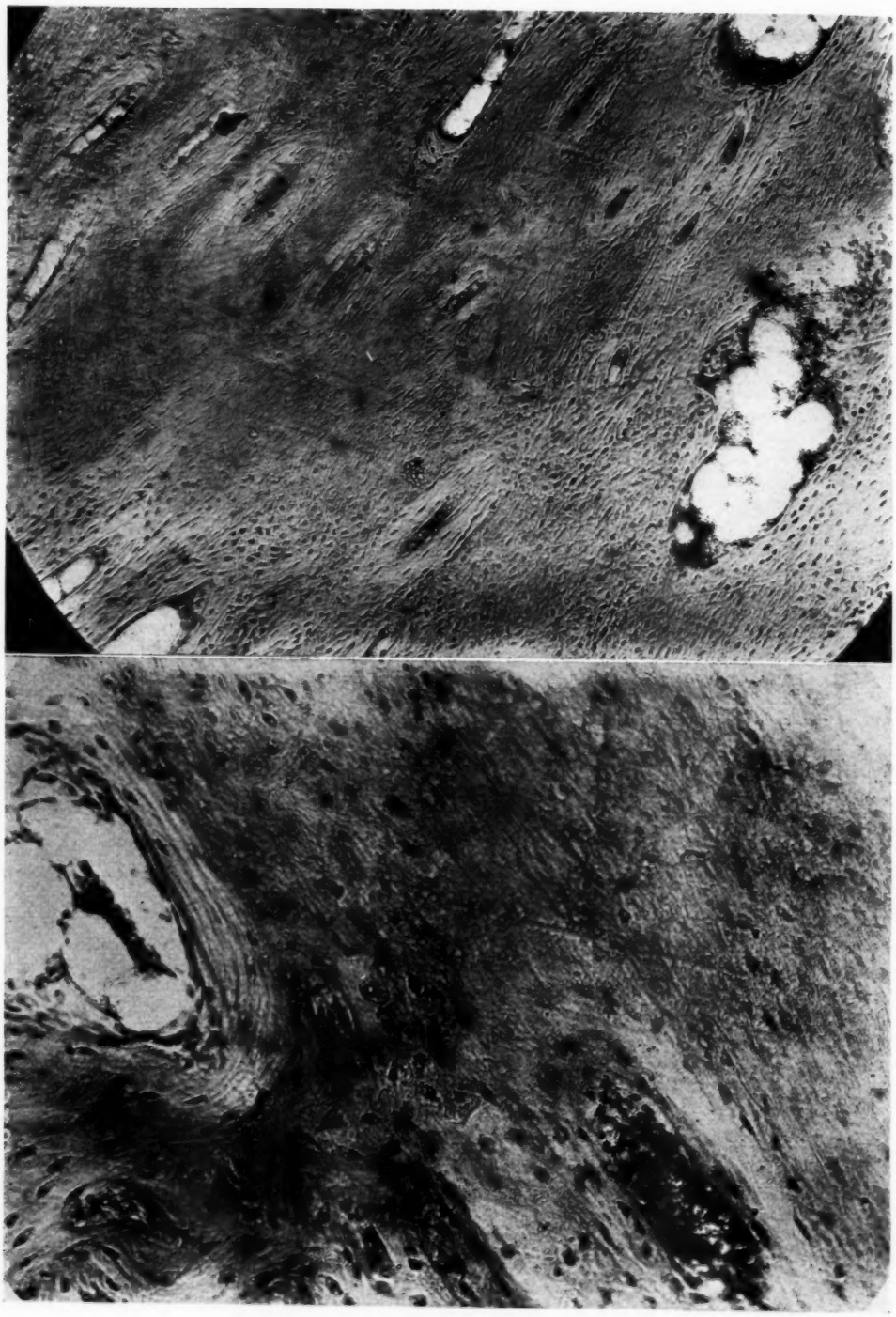

Fig. 9 (*above*). One of included fragments of Figure 8 (× 150).
Fig. 10 (*below*). High power of section of Figure 9 (× 375).

the features noted in the last figure. The background again is the loosely meshed tissue of growing fibroblasts with its accompanying capillaries—granulation tissue. In it is the lace-like mesh of calcium deposition. Any single mesh is found to consist of a central capillary surrounded by an area of fibroblasts and the deposition of a rough ring of calcium outside this area —that is, in the relatively avascular zone. The cells lying within the calcium deposition again are identical with those comprising the areolar tissue outside, and the areas of calcium deposition merge very gradually into the matrix of the areolar tissue wherever they do not link up with other areas of calcium deposit. (A) represents a capillary, (C) the areolar tissue which surrounds the capillaries, and (B) the deposition of calcium about the cells in the outer zones of (C)—the relatively avascular areas.

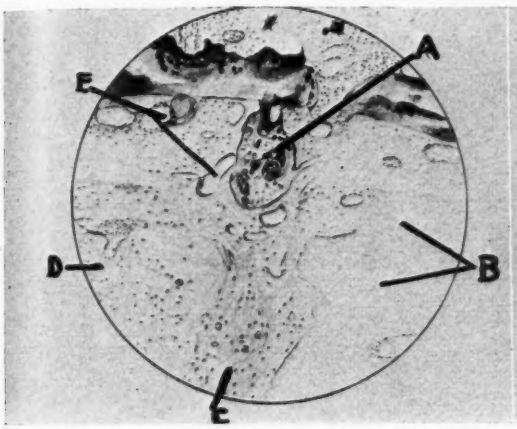

Fig. 11. *A*. Cartilage. *B*. Adjacent cortex. *C*. Callus. *D*. Normal cortex. *E*. "Cyst" formation.

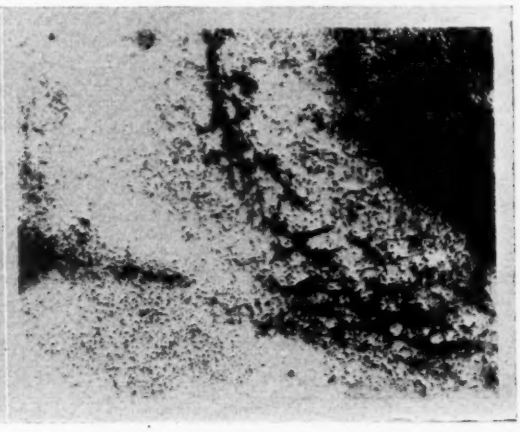

Fig. 12. Three-day human callus.

Figure 14 is a high power microphotograph of the preceding one, designed to bring out the details. One can see clearly the identity in morphology of the fibroblasts and the bone cells, the background of loose meshed fibroblastic areolar tissue and the mode of formation with the central capillaries, the surrounding areolar tissue and the calcium deposition in its outer relatively avascular zones. Also clearly demonstrated is the gradual merging of the calcium-impregnated areas with the collagen background of the areolar tissue, so that in spots it is difficult to tell just where calcium begins and ends. One is struck by the fact, after the study of many of these sections, that if this were a water color and it were possible to wash out the deep pink of calcium the resultant picture would be the classical one of granulation tissue, although no morphological change would have been effected. Between this early bone and granulation tissue there is no appreciable difference save that some process has deposited calcium in a constant pattern throughout the granulation tissue. How is this done? Is this a cell activity or a purely physico-chemical process? Where does the calcium come from and why does it deposit as it does?

Figure 15 is a drawing representing the appearance of a microphotograph of callus appearing in muscle adjacent to but not connected with a fracture of a human humerus of twelve days' duration. It was recovered at operation. There are several points of significance in it. In the first place, this new bone formation has occurred in muscle tissue unconnected with bone. Bone has appeared without a bone nidus for growth, unless we are to premise the wandering of osteoblasts from the fracture site to the site of bone formation in the muscle. Without that premise we must assume a local metamorphosis of some other cell into a specific bone-forming cell, or a process which occurs without the action of any functionally specific cell. In the second place, we must consider where the calcium comes from which is utilized to form this new bone and how it is utilized. Morphologically one notes in this section all the features seen in previous sections, including the diversity of tissue present, the morphological identity of the cells, the typical arrangement of the new-formed bone with its central vascular channels (B) surrounded by areolar tissue and, in the avascular zone, the newly deposited calcium (C). The degenerating muscle fibers are seen at (A).

Figure 16 gives another example of early callus. It shows again the typical arrangement of the new-forming bone and, in addition, emphasizes the apparent diversity of tissues present and the extremely gradual merging of the one into the other. It emphasizes the point that these tissues at this stage are not to be differentiated by cellular morphology, but by the differences in background. Here we find, with practically identical cell types morphologically, tissues which we can call bone, hyaline cartilage, fibrocartilage and fibrillar connective tissue, purely because of the ground substance in which the cells lie. These ground sub-

stances are or are not the product of the cells. They vary, although the cells show no visible evidence of functional variation. Moreover, these backgrounds merge very gradually one into the other. Is this evidence of varying degrees of functional metamorphosis finally become so-called scar tissue with long flattened cells lying between the collagen fibrils comprising the ground substance.

Figure 19 represents a microphotograph of the callus adjoining one of the bone ends at a fracture site. This

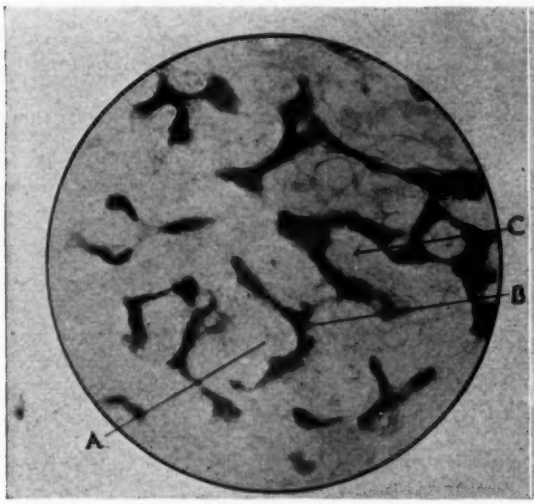

Fig. 13. Nine-day human callus.

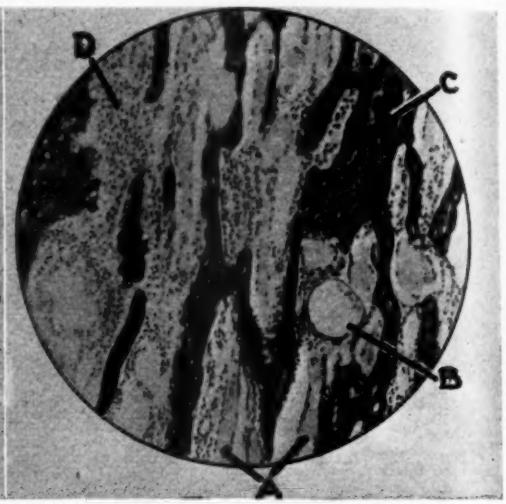

Fig. 15. Human callus, 12 days' duration. *A.* Degenerating muscle fibers. *B.* Blood vessel. *C.* Calcium. *D.* Areolar tissue.

of cells without morphological change? Or is it evidence of the lack of specific connection between the functioning cell and the ground substance in which it is embedded?

Figure 17, a microphotograph of another specimen of early callus, again focuses attention on all the points cited in the discussion of the preceding figure. These specimens of early callus are well worth careful and detailed study as to all the points cited in connection with them.

Figure 18 shows a specimen of callus of later date than shown in the preceding illustrations. The formation of the Haversian canal systems in spongy new bone is now apparent. The avascular zones are the sites of the intercommunicating bands of calcium deposition. These bands are becoming more sharply defined. The gradual merging of the ground substance of bone and connective tissue, although still present in numerous areas, is becoming a less marked feature. The calcium bands seem in general to have become more sharply defined and to have definite sharp margins. Cell morphology is still generally undifferentiated, although it is becoming apparent that those cells which lie in the denser and more rigid medium, which is becoming constantly more dense and compact because of continued calcium deposition, are beginning to look flattened out and elongated. Casting about for comparison, one calls to mind that in loosely meshed new growing connective tissue in a healing process one observes an identical change as that tissue becomes denser and denser, to is a late stage in the process. Again we see the characteristic pattern of the new bone formation, heretofore commented upon; again the diversity of tissue types. In the lower left quarter of the picture the merging of one background into the other is apparent. Elsewhere the definition is sharper. Morphological differentiation of the cells of new bone and connective tissue is difficult. Where that new bone is densest, there the cells are perhaps a little more flattened and elongated. When we look at the old very dense cortical bone, we note that the cells are elongated and flat and are therefore morphologically different from those of the newformed bone. Does this morphological change mean functional difference or is it purely the physical effect of the embedding medium? Note also in this figure the evidences of some death in the old bone cortex as evidenced by the absence of nuclear staining and the empty cell spaces. Note also that the new bone is formed about this cortical bone as a nidus and not down in the left lower quarter, where no preëxisting bone is present. Is the cortex a nidus about which the new bone forms because of the presence there of live bone cells which take part in or initiate the process, or because of the presence of the dead bone? How can the dead bone act as a nidus for new bone formation?

In *Figure 20* we have a microphotograph of tissue taken from between the bone ends of a fractured human femur at the end of twenty-eight days. There was considerable motion at the site of fracture. The diversity of tissue present is again apparent, as also is the

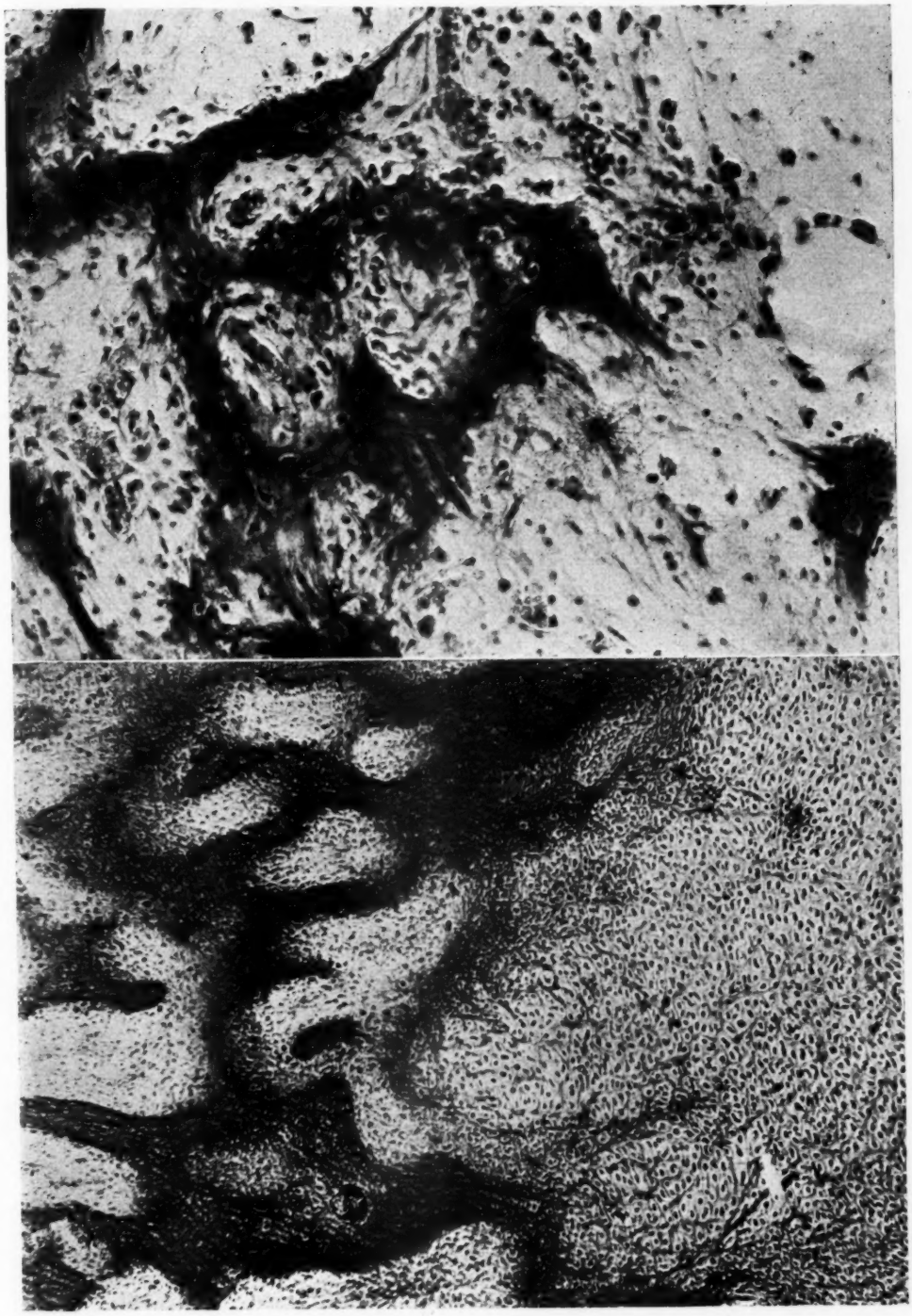

Fig. 14 (*above*). Higher power of Figure 13.

Fig. 16 (*below*). Early callus.

pattern common to new bone to which attention has been called in previous figures. In a low power picture, such as this, cellular morphology cannot be the object of accurate observation. One notes readily, however, a larger amount than usual of cartilage. Whence is this tissue derived and how? If morphology is to count for much, surely we should suspect this tissue of specificity if we are going to talk of specific osteoblasts *per se* or by adaptation or metamorphosis. Frequently repeated one meets the assertion that undue movement at the site of a fracture favors the presence of cartilage in the callus. What is it that such mobility at a fracture site can influence to have such effect, if this story be true?

Having viewed the observed facts as to what actually happens and propounded some of the questions which these observations have brought into our minds, let us go now into the question as to how this process which we have studied is accomplished.

We have been accustomed for years to talk of osteoblasts. These we have conceived to be specialized forms of connective tissue cells—cells having the specific function of forming bone by the laying down of calcium salts (?) about them in the intercellular substance in which they happen to be. Assuming for the moment that this idea is a correct one and that we are on safe ground, let us follow out the story. At the time of fracture we have seen that there is hemorrhage from the bone ends and in the traumatized soft parts, with some tissue death in both those structures. As a result of the effused blood (which is a marked chemical irritant when free in the tissues), of the physical insult, and of the mechanical blocking and destruction of both blood and lymph channels, there is added a profusion of exudate rich in fibrin. The necessary ferments being at hand, set free or activated by tissue death, the hemorrhage and exudate clot. That is, they lay down an entangling fibrin network between and about the bone ends and in the adjacent soft parts to which they have access. The remaining fluid portion of the blood and exudate follows tissue planes to the surface to appear eventually as the extensive ecchymosis seen in fracture cases. The fibrin clot is promptly organized as granulation tissue by the ingrowth of capillary buds and their surrounding fibroblasts. This tissue is derived solely from the connective tissue in the structures involved—periosteum, endosteum, vessels, fasciæ, the stroma of muscle tissue and the areolar tissue between them. Experimentally, as we have seen, it is possible to prove that under given conditions most of this may come from soft parts without the bone. In a subperiosteal fracture it must obviously be derived totally from the soft parts of the bone—periosteum, so-called endosteum, and the areolar tissue of the marrow spaces. This granulation tissue, from wherever derived, surrounds the bone ends and joins them one with another and with the adjacent and available soft parts. *It constitutes the healing of the wound in bone,* just as similar tissue constitutes the healing process in muscle, cornea, liver or other tissue. Up to this point we have been talking of observed facts. Assuming the osteoblast as an identity, we may now theorize as to what happens to cause calcium deposition in the manner in which we have seen it deposited to form a calcium matrix granulation tissue called bone instead of a collagen matrix connective tissue. In this granulation tissue must be enmeshed such osteoblasts as were freed without death by the mechanical violence of the fracture rupturing the cell spaces. A certain number of osteoblasts are killed by the violence plus the inflammatory reaction, and the bone matrix occupied by them is autolyzed by ferments freed by tissue death and is partly absorbed but mostly remains as a high calcium concentration locally in the tissue fluids. The fate of this matrix is also an observation of fact and not theory. The exact state in which this calcium exists is unknown. The exact state in which the calcium of the blood exists is not known. Both these points will be referred to again. To return to our osteoblastic muttons, other osteoblasts grow out from the ends of the bone, invading the granulation tissue and laying down their calcium matrix about them as they do so in increasing density. The very early callus of thick gelatinous fibrin-rich exudate invaded by granulation tissue with relatively little calcium becomes harder and firmer, but completely retains its form of granulation tissue. Morphologically there is no way of telling these osteoblasts from the cells of connective tissue, or the granulation tissue from the early callus, as we have seen, except by the presence of a calcium deposition which merges imperceptibly into the collagen of the connective tissue fibrils. In many of the experimental specimens actual fibrillar striations are seen directly continuous with those of adjacent connective tissue. The calcium involved must be part of the cells' metabolic activity, and

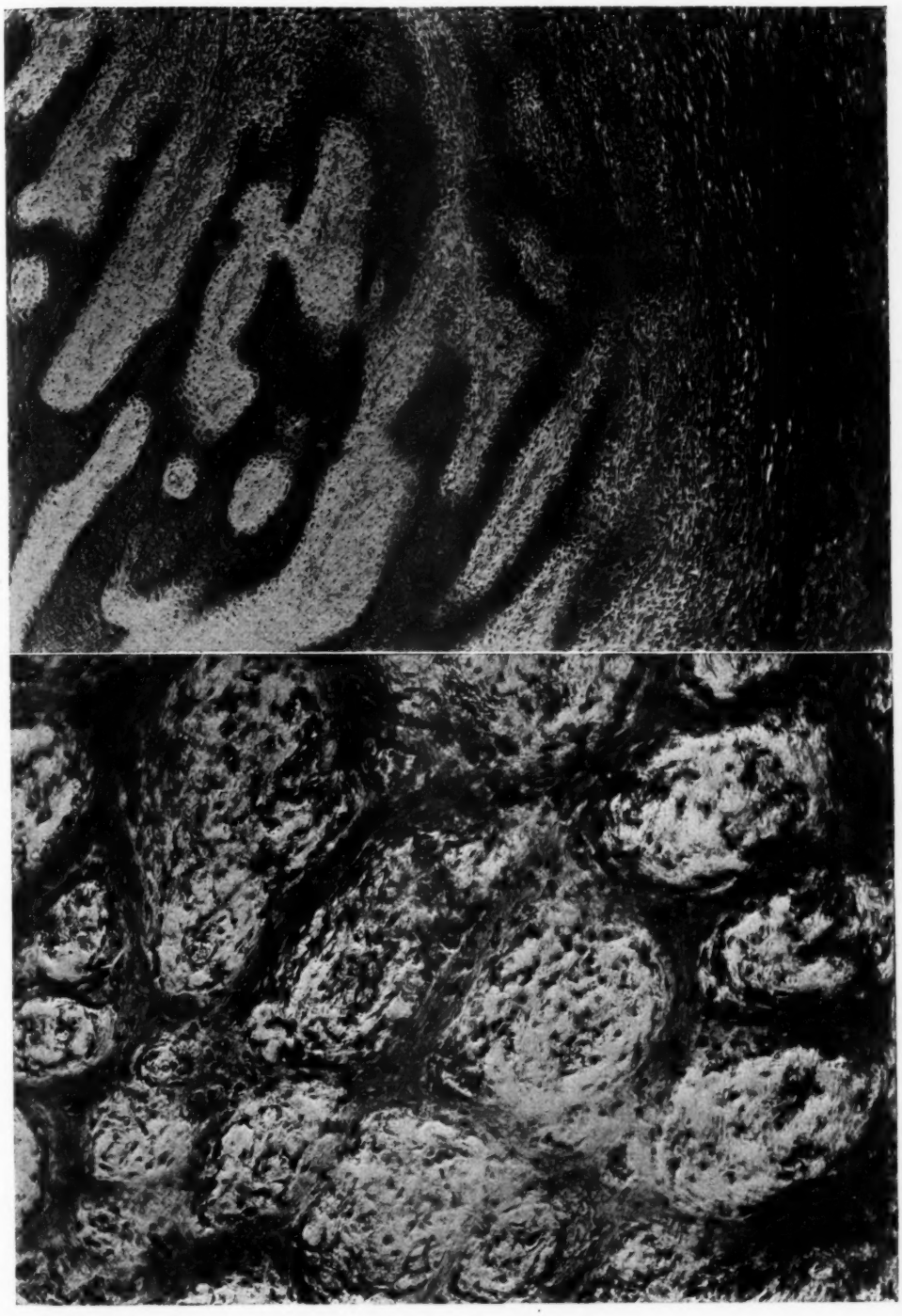

Fig. 17 (*above*). Early callus. Compare Figure 16.

Fig. 18 (*below*). Late callus. Compare Figures 16 and 17.

its source should logically be, and has been presumed to be, the blood stream. The argument as to the exact source of these specific osteoblastic cells, whether from periosteum's cambium layer, bone, or endosteum—which has been in progress since the days of Barth,[8, 9] Ollier,[10] Duhamel[11] and John Hunter,[12] through MacEwen[13] to modern times—can be epitomized in the statement that under the osteoblastic theory from today's viewpoint the cells are derived from all three of the sources mentioned.

Bony callus having been formed thus, the molding or architectural story from then on is a physical one and has no bearing on the formation of bone.

Here we have what sounds like a pretty straightforward story. Yet there are numerous flaws in it. Sometimes the activity of these specific cells is apparently interfered with. The purely mechanical bar of interposed tissue or widely separated fragments we can, of course, dismiss as readily understandable, no matter what idea we may have as to the mechanism of bone repair. But frequently under ideal conditions—that is, with early and accurate reduction and treatment thoroughly approved by textbook standards—we are confronted, perhaps, by an exceptionally slow callus and bone formation which we call delayed union. Or we get an absolute failure of callus or bone formation which we call a non-union. Or, still again, we get good callus, satisfactory *x*-ray evidence of bony union with solidity of the parts, only to find absorptive changes coming on with marked decalcification at the fracture site and eventually a fibrous union remaining. This is not an unusual story in fractured neck of the femur, the proximal neck fragment absorbing after as long as six to nine months right down to the intertrochanteric line. Under any of these circumstances we are given to stimulating these specific bone-forming cells in various ways. Empirically it has been discovered that they can at times be awakened from their unexplainable apathy by various forms of insult. When so aroused they seem to proceed to lay down calcium, as a well-behaved osteoblast should do, and bony union occurs. At other times such insult seems to be of no avail, no bony union occurs and we are left with a so-called non-union. Some of the physical means of stimulation used are frequently repeated tappings over the fracture site with a light mallet, repeated grinding together of apposed surfaces with moderate force and only slight motion range, repeated needling of the fracture site, exposure to *x*-ray and weight-bearing with support. The chemical means include the injection at the fracture site of numerous irritants from blood through a long range of chemicals. One might feel, perhaps, that these cells failed because the source for their calcium metabolism, *viz.*, the blood stream, was deficient in amount or in kind. Feeding these patients calcium or phosphorus or both has no appreciable effect, nor in my own experience has the feeding of vigantol or irradiated ergosterol, with the idea of increasing the blood phosphorus, or of parathyroid, with the same idea in mind. Some years ago Tisdale[14] reported the consistent finding of a raised blood phosphorus in adults during fracture healing extending over a period of weeks, and construed the phosphorus rise as an indicator of increased calcium activity. The absence of such a rise was presumptive evidence of a threatened non-union. He found such a conjunction in a few cases. Subsequent workers have consistently failed to check on his findings. Working with Eddy,[15] the bio-chemist at Teachers College in New York, on a series of cases in New York Hospital and other hospitals, I could find only partial corroboration of Tisdale's conclusions, but found that the same phosphorus rise occurred in any extensive soft part healing—such as large granulating operative wounds, extensive granulating burns and extensive fibrosis due to phthisis. Also, the extent and amount of callus formation bore no definite relation to the phosphorus rise. Eddy's impressions were not in exact accord with this opinion. His chief interest lay in the methods used to determine the blood phosphorus figures—mine in the clinical study of the cases. To date there is no corroboration over a year's period in the Fracture Service at the Presbyterian Hospital in New York, where a blood phosphorus and blood calcium is taken weekly on every fracture case. Cases showing actual decalcification at the site of fracture have shown normal calcium and phosphorus blood findings. This is true also of fractured neck of the femur cases. It seems difficult, then, to blame non-union on lack of blood calcium to provide to the osteoblasts. By some of those who hold to the osteoblastic theory the problem is now being worked on, under many technical difficulties, on the basis of

Fig. 19 (*above*). Late callus about broken end of bone.

Fig. 20 (*below*). Fractured human femur, 28 days. Considerable motion at fracture site. Tissue removed at operation from between bone ends.

the relative proportions of diffusible and non-diffusible calcium present in the blood stream.

Having considered these points, another series of observed facts appears which are upsetting. If we are to regard osteoblasts as specific, certainly we must so regard cartilage cells. At the site of fracture in the shaft of an adult long bone there is surely no cartilage, and yet callus in its formation, as one can see from our sections, contains bone laid down both in cartilage and in connective tissue. More than that, the bone form of matrix with the cartilage type of cell exists in callus—an association hard to correlate with a specific osteoblast, since morphologically no cells even remotely resembling osteoblasts are present, and yet bone is present. We call to mind also the lack of any distinctive cell type in many of these sections. The cell type has apparently little correlation to the type of intercellular substance.

At different times we note active bone formation in tissues far removed from bone as the result of injury, disease, or chronic irritation. Under the microscope this is morphologically real bone with Haversian canals, not a mere calcification. It is found in chronically inflamed and fibrotic tonsils, in abdominal scars, and in the late stages of arteriosclerosis in the outer walls of the vessels where run the vasa vasorum. It is also found following trauma to muscles, as the so-called traumatic myositis ossificans (in the true case it has no connection with bone), and it is found in the form of rider's bone. Experimentally it is produced by numerous methods. If we tie off the intrinsic circulation of a rabbit's kidney, leaving only the capsular supply with or without omental aid, we will get true bone formed in a certain percentage of cases. Neuhof[16] has secured it in fascia lata transplants into the bladder wall. The only constant factor in all these instances of abnormally situated bone formation is a circulatory disturbance, with some death of tissue. If we hold to a specific osteoblast we must then believe that these abnormal sites for bone production are the results of the activities of osteoblastic rests of fetal origin, or that under certain indefinite conditions of which we know only two constants—circulatory disturbance and slow tissue death—ordinary fibrillar connective tissue cells may by metamorphosis become osteoblastic in function. The why and wherefore of that metamorphosis is beyond our ken.

To go back to our non-union for a moment. When other means fail, Bier's hyperemia sometimes succeeds—this is essentially the introduction of an artificially produced vascular disturbance into the picture—and, just as in tying off the vessels of a rabbit's kidney, sometimes it works and sometimes it does not. Nor have we any means of telling just which result is going to be effected in any given case. When all other means fail we are driven to operative procedure, the most effective method being some form of bone graft. If we hold to a specific osteoblast or an oseteoblast by metamorphosis it stands to reason that we must believe we are transplanting live osteoblasts, or are creating conditions favorable for the metamorphosis of osteoblasts from connective tissue cells, or both. Some things we know we do by operating—we kill some bone by trauma, we invade the soft parts which in healing produce granulation tissue and we change the circulatory status.

We can now take up some of the more recent observations on the bio-chemistry of this process. The work of Wells,[17, 18] Howland,[19, 20] and others has established many apparent facts in connection with the blood and tissue calcium and phosphorous content in health and disease. It has also been shown that hyaline cartilage in the presence of calcium inorganic salts shows a marked avidity for the adsorption of these salts. It has been shown also that under proper P^H conditions these calcium salts can be caused to precipitate or deposit in the hyaline matrix. Watts[21, 22] has shown that the form in which calcium exists in bone, a homogeneous mass, is entirely different from the form in which calcium appears when precipitated from solution *in vitro*. By alterations in the P^H it has been observed also that calcium in colloid combination can be deposited from that combination without any cellular activity. Bassett[23] has indicated a complicated organic calcium combination of large molecular size, which contains calcium and phosphorus with carbonate attached as CO_2 and which he calls hydroxyapatite. He has shown that this compound is capable of stability in both blood and tissues. Robison,[24, 25] of England, has produced evidence to show that in calcium compounds showing this same adsorption into

cartilage the precipitation of the calcium in the cartilage may be induced by the activity of a phosphatase or esterase which by splitting a hexosephosphoric ester frees phosphoric acid and may therefore bind the calcium. Moreover, it is conceivable that by alterations in the P^H of the medium in which this reaction takes place, it can be slowed up or stopped. All ferments, as we know, are active to their full extent only in the presence of a certain P^H. Altering that P^H will slow up the ferment activity or even stop it, so that the normal absorption of calcium which constantly goes on under those circumstances might even exceed the amount of calcium deposition and decalcification of bone might occur. Not infrequently we clinically observe such decalcification—here is a much more logical and simple explanation of it than any that can be given under our osteoblastic theory of bone origin.

Considering all these factors, it becomes apparent that if we are to disregard cell activity we have to consider what could vary the local P^H in order to explain failure of calcification, slowness of calcification, normal calcification and even decalcification. It becomes apparent that we have a single explanation of all these phenomena within our grasp. As we have pointed out, the one constant factor in the production of bone by injury, disease or experiment is circulatory change. We have seen also that the one constant factor in the recognized forms of treatment for delayed and non-union has been circulatory change. It is logical to feel that both qualitative and quantitative local blood supply changes will induce local P^H changes. Experimentally this has been shown to be true (by the use of quinhydron or hydrogen electrodes in the tissues and a string galvanometer as a potentiometer to record changes in potential) in acute inflammatory processes in the soft parts. It requires little change in P^H, however, to affect ferment activity, and the slight changes necessary are unmeasurable accurately in the tissue fluids by our present means because of the range of instrumental error, as well as the discrepancies due to purely physical causes such as muscular contractions, circulation, etc. We can say, however, with reasonable certainty, that quantitative and qualitative change in the circulation locally very possibly may induce local changes in P^H, even when slight in extent. Here, then, may lie one phase of our story. Dependent upon the circulatory status of the soft parts, in the bone and in the adjacent tissues as well, will depend the P^H of the granulation tissue produced from those soft parts. On that P^H will depend whether ferment activity is normal, slow or absent—that is, whether calcium deposition is normal, giving union; or slow, giving delayed union (if slightly in excess of the normal absorption process which is constantly going on) or non-union (if it just about keeps pace with this absorption); or whether actual decalcification occurs because there is no calcium deposition occurring to offset even the normal absorption process in the bone. The ferment production may be the result of tissue death—and results wherever a certain amount of connective tissue death occurs.

Why, then, if the blood calcium and phosphorus are normal, does not calcium deposition as bone occur more frequently where there is tissue death with circulatory disturbance?

On this point I will cite work[7] which is now in progress, in so far as it has gone.

If we resect 2.5 cm. of a dog's radius and throw it away, leaving the gap in bone to heal as it will, we get a fibrous union filling the gap between the two bone ends with a little ossification just adjacent to the bone ends where we have killed some little bone by our operative procedure.

If, instead of discarding the resected portion, we remove all periosteum and endosteum and break it up into chips and place *them* in the gap, we get a solid bony union, although, as we have seen from our sections, these bone chips are dead and are autolyzed, at least in part.

If, to make sure of their death, we boil the fragments before putting them back, we get bony union notwithstanding.

If we decalcify these fragments before we put them back, *we get no evidence of bony union.*

Obviously, viability being disposed of as a factor, and everything else except the calcium being disposed of as a factor, the rôle of these bone chips seems to be to supply calcium for the calcification of the granulation tissue constituting the healing process.

Therefore, I have resected in other dogs the same amount of radius, discarded the fragment and placed in the 2.5 cm. gap powdered sterile $CaCO_3$ and $Ca_2(PO_4)_3$—supplying an artificial source of calcium in the gap previously filled by bone—and *solid bony union has resulted.* Similar

procedure using sterile powdered calcium hexose phosphate has again given solid bone in the 2.5 cm. gap.

If, before putting in the calcium, some of the dog's blood was defibrinated and the fibrin placed in the gap, the union was more rapid. As control animals with merely the fibrin placed between the fragments gave fibrous union, we can conclude that the rôle of the fibrin is merely to aid in the more rapid organization by granulation tissue. It is possible also that the fibrin has a similar affinity for calcium to that of the hyaline of cartilage. The hyaline of cartilage and the collagen of new-formed connective tissue are very closely allied chemically, and such affinity in collagen has been shown to exist. Such an affinity in the fibrin would tend to hold the calcium for local use and to prevent its dispersion by the local circulation.

Here, then, we may have good logical reason for the occurrence of these delayed unions, nonunions and decalcifications, even in the presence of normal P and Ca. The calcium source for calcification of the granulation tissue is local and is the dead bone at the site of fracture, autolyzed by ferments set free or activated by tissue death and held in situ by the affinity of fibrin (?) and the collagen of granulation tissue and deposited in that granulation tissue to form bone by another ferment activity, that of a phosphatase being used purely as an example. The vital process would then be ferment activity in the presence of a local calcium source, and the completeness or failure of the process would depend upon the local P^H of the tissue fluids induced by the local circulatory status. On this basis bone formation would not occur in soft parts where tissue death and circulatory disturbance co-exist unless there were a local concentration of calcium at the site (regardless of what the blood phosphorus or calcium might be) and unless the circulatory change were such as to produce proper tissue fluid P^H conditions. The factors which might cause local concentration of calcium in an isolated soft part region, thereby providing the local calcium source, remain to be determined. It has been shown that the growth of certain bacteria in the soft parts can induce such a local calcium concentration. This and other factors capable of doing the same thing are under investigation.

The possible conclusions we may draw from the material herein presented are as follows:

1. Fractures always heal, unless there is a mechanical bar to tissue growth.

2. The healing is accomplished as with wounds elsewhere by the growth of granulation tissue.

3. This granulation tissue is derived from the wounded soft parts of the bone—periosteum, endosteum—and the areolar tissue about the vessels in the marrow cavity and elsewhere. If there is tearing of the periosteum it is derived from the soft parts about the bone as well, in proportion to the access allowed them to the fracture site. Sometimes the major part of this granulation tissue comes from the soft parts about the bone.

4. Delayed union is a failure of the healing tissue to calcify at the average rate.

5. Non-union is a failure of the healing tissue to calcify at all.

6. The source of calcium for this calcification is dead and autolyzed bone at the site of fracture and not the blood stream.

7. Vascular changes probably induce local tissue P^H changes which may strongly influence precipitation of calcium salts from solution or colloid combination or ferment activities on large radicles of organic calcium salts—such as hydroxyapatite or calcium hexose phosphate.

8. Given tissue death, a local calcium source, a vascular status producing the proper P_h, and granulation tissue, bone will form without the presence of any bone cell.

9. Bone as a calcium source can be experimentally replaced by both inorganic and organic salts of calcium (hexose phosphate). Either precipitation or ferment splitting activity may be responsible for the calcium deposition; the latter seems the more probable.

10. Bone and soft part circulation are of prime importance in the process of calcification of fracture healing and should, therefore, be prime objects of attention *early in the bone healing process.* The time to use elevation, heat, massage, diathermy and active exercise is at that time, if possible; it is much more important than later in the course.

11. Mouth administration of Ca, P and similar drugs is ineffective. If calcium is needed it must be supplied locally and not via the blood

stream in any individual whose general calcium metabolism is normal.

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SYMPTOMS AND COMPLICATIONS OF CHRONIC URINARY OBSTRUCTION*

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STRANGE as it may seem, one of the commonest human ailments is also one that is frequently overlooked; namely, hypertrophy of the prostate gland. One would naturally think that a condition so easily diagnosed on rectal examination would scarcely ever be missed. Failure of diagnosis occurs, in most cases, either because of the absence of any urinary symptoms, or because the prostatic obstruction is confined to the intravesical portion of the gland so that rectal examination gives negative results.

One would infer that in cases in which the prostatic obstruction is sufficient to cause considerable retention of urine, the patient would be aware of some urinary symptoms which would call attention to the condition. However, this is frequently not the case, since it is not at all uncommon to find residual urine to the amount of 500 c.c. or more without the patient being aware of its presence, and with no urinary disturbance noticeable to the patient. Probably the increase in residual urine is so slow and insidious that the patient is not aware of any additional discomfort. It is surprising how the human system will accommodate itself to the presence of a large amount of residual urine and how all other functions proceed as regularly as though nothing had happened. Apparently, however, there is a limit to the amount of urine which can be retained in the bladder without causing disturbance of renal function. This limit probably varies considerably in different persons, depending on a variety of factors, chief of which probably is the degree of protection against back-pressure offered by ureteral peristalsis and the ureterovesical mechanism. When this barrier has been broken down and the function of the kidneys is interfered with, symptoms arise which are not referred to the urinary tract but which are included in the wide range of symptoms accompanying renal insufficiency.

Among the secondary symptoms more commonly observed are anorexia, nausea, vomiting, headache, dizziness, loss of weight, general weakness, and other signs of constitutional disturbance. Therefore, when a male patient aged more than forty years comes to the physician with headache, loss of appetite, occasional nausea or even vomiting, with pain referred to the epigastrium, or with such symptoms as backache and gradual loss of weight, it behooves him to make a rectal examination in the course of the general examination, and to determine whether there is any evidence of prostatic enlargement or other evidence of urinary retention. The complete medical examination in every case, and particularly in the adult male, should include a rectal examination, and unless this is made as a routine, many clinical conditions will remain undiscovered. It is curious that there should be an apparent prejudice against rectal examination, when by it data are so easily discovered which may be of such fundamental importance in ascertaining the cause of symptoms.

It is not at all uncommon in my experience to observe patients concerning whom a previous clinical diagnosis of nephritis has been made and in whose cases the results of general examination and the laboratory data suggest advanced nephritis. Many of these patients have been placed on antinephritic treatment, including dietary precautions, for months before the true condition is recognized. The similarity between the clinical data observed in cases of nephritis and those in renal insufficiency resulting from urinary retention, often is striking. Anorexia, nausea, loss of weight, weakness, headache and dizziness are common to both conditions. The data obtained on urinalysis are usually similar. The total output of urine in twenty-four hours is usually more than 2,000 c.c. in both conditions, and the specific gravity is usually low, averaging from 1.006 to 1.010. The excretion of phenolsulphonephthalein in two hours is often

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only a trace, with marked delay in time of appearance. As estimated, the blood urea and blood creatinine are usually markedly elevated; of the former often there are more than 100 mg. and sometimes more than 200 mg. for each 100 c.c. The blood pressure is usually markedly elevated; a systolic pressure of 200 mm. of mercury is not an uncommon occurrence.

Among the most valuable methods of clinical differentiation is the ophthalmoscopic examination. Characteristic retinal changes would almost necessarily be present with nephritis sufficiently advanced to cause clinical signs, such as those described. However, when urinary retention is the underlying factor, marked retinal changes are usually absent.

As an illustration of this differentiation may be mentioned a case which came under my observation not long ago. In the course of general examination the examining physician found clinical data which led him to believe that the condition was one of nephritis. An ophthalmologist was called in to make a retinal examination and when he had finished said, "This patient does not have nephritis; he has enlargement of the prostate gland." He arrived at the conclusion purely by differentiation, realizing that a patient with clinical evidence of advanced nephritis, and with normal eyegrounds, could hardly have anything else than urinary retention to account for his condition.

An interesting feature in the differentiation of nephritis and the insufficiency of renal back-pressure is the frequent absence of parallelism in renal back-pressure between the retention and excretory renal functional tests. As a result of interference with renal drainage caused by back-pressure, the excretion of dyes, such as phenolsulphonephthalein, is greatly diminished. It is not uncommon to find that only a trace of phenolsulphonephthalein has been excreted two hours after its intravenous injection, giving the impression of widespread renal destruction. On the other hand, retention tests may show that the blood urea is only moderately elevated and the blood creatinine may be practically normal. In contrast, there is usually closer parallelism between the excretory and retention tests in nephritis. If, in a case of evident renal insufficiency, there is any question as to the predominant etiologic factor, differentiation can be made by renal functional tests following drainage of the urinary tract. If the renal insufficiency is temporary and is caused by urinary retention, the laboratory data will undergo a great change when drainage is reëstablished. First to improve will be the blood urea, which may drop 30 or 40 mg. within a week. The excretion of phenolsulphonephthalein gradually will improve, and in the course of several weeks will approximate normal. How different the picture is with true nephritis or when the kidneys are permanently injured. It is then only with great difficulty that the blood urea can be reduced, and the excretion of phenolsulphonephthalein may remain but slightly improved, in spite of every effort.

It is strange how frequently patients present themselves with symptoms referable to the alimentary tract entirely. It is not uncommon for patients to give a history which can easily be regarded as typical of gastric carcinoma, such as vomiting, epigastric pain, loss of weight and cachexia, all of which can be explained by renal insufficiency secondary to urinary retention. It is curious that many patients with prostatic hypertrophy will also complain of considerable constipation. Whether the enlargement sometimes acts as a mechanical factor in the rectum or whether it is a reflex phenomenon from irritation in the prostatic region is difficult to determine.

The physical evidence of urinary retention other than that which may be obtained from prostatic enlargement must be considered. One might think that an overdistended bladder could be made out rather easily on abdominal palpation. However, this often is not the case. It is surprising to what extent the bladder may be distended and still be difficult to discover on either abdominal or rectal palpation, or by percussion. When there is an evident abdominal tumor in the median suprapubic region, one must, of course, infer vesical overdistention, bearing in mind, however, the other abdominal conditions which may cause tumor similarly situated. The bladder, however, does not always distend uniformly, so as to cause a symmetric tumor in the median suprapubic region. In fact, it happens frequently that there is unilateral enlargement of the bladder, for half of the musculature of the bladder may give way and that half may be-

come distended, while the remaining portion of the musculature of the bladder hypertrophies. As a result of this, the tumor may be felt entirely in either the right or left lower abdominal quadrant. I distinctly recall a case of this kind in which a surgeon had felt a cystic mass in the lower right quadrant of the abdomen and had advised immediate operation for abdominal tumor. Although the patient did not have urinary symptoms and although on rectal examination there was little, if any, prostatic enlargement, the tumor disappeared on introduction of a catheter into the bladder.

One might expect that examination of the urine would call attention to the presence of urinary retention. However, in many cases of retention, urinalysis gives negative results or reveals, at most, occasional leukocytes or erythrocytes. Needless to say, the presence of even a few cells should always lead to further investigation of the urinary tract. With prolonged urinary retention of marked degree, there is usually definite reduction in specific gravity, which also might call attention to possible involvement of the urinary tract.

Of greater value would be the evidence obtained by renal functional tests. As the result of prolonged renal back-pressure there is often excretion of only a trace, or an extremely low percentage, of phenolsulphonephthalein, and with the low excretion of dye the blood urea is usually distinctly higher than normal. There is lack of parallelism between the retention and the excretory tests because the amount of the dye excreted is not a true index of the secretory power of the kidney. Because of the back-pressure of the retained urine there is failure of excretion of the dye rather than failure of secretion. Frequently, the blood urea is raised only to a moderate degree, whereas the excretion of phenolsulphonephthalein is markedly reduced.

It must be emphasized that urinary retention cannot be excluded because evidence of prostatic enlargement is not felt by rectum. The following conditions may be present even though rectal examination gives negative results: (1) prostatic obstruction confined to the intravesical or median portion of the gland; (2) disturbance of innervation of the bladder, usually resulting from disease of the central nervous system, or (3) diverticulum of the bladder.

It is frequently surprising how much prostatic obstruction may be present in the intravesical portion of the gland in cases in which rectal examination reveals little, if any, enlargement. Such obstruction can be determined by means of cystoscopic examination only. The presence of retention in such cases may be surmised from the data previously described, and in case of doubt can be verified by catheterization. The degree of urinary retention is not commensurate with the degree of prostatic enlargement felt by rectum. Frequently, the prostate gland is comparatively small and still is so situated that the obstruction is great. Since the widespread use of the cysto-urethroscope, cicatricial contracture of the vesical neck also is being recognized as a common cause of urinary obstruction.

Another form of prostatic enlargement which has not been mentioned is prostatic carcinoma. This condition may be the cause of a variety of general symptoms without any subjective evidence of urinary obstruction. Chief among these is pain referred to various portions of the body. One of the commonest causes of so-called lumbago, sciatica or neuritis referred to the sacral or lumbar region or to either leg, is that caused by prostatic carcinoma. Many patients have been observed who have complained of pain referred to these regions, and of loss in weight or strength, but in whom the prostatic condition has been neglected because of absence of urinary symptoms. The abnormality of the prostate gland could be discovered only by rectal examination as a routine. It hardly would be necessary to describe the physical characteristics of prostatic carcinoma, since all are familiar with the peculiarly firm, nodular character of the gland.

Urinary retention resulting from disturbance of innervation of the bladder is common and may easily be overlooked. Such retention may be caused by disease in the central nervous system or by terminal neuritis. Although the former condition usually is accompanied by clinical evidence which would call attention to a lesion in the central nervous system, such data are often obscure, and in the early stages of the disease may be absent. On the other hand, retention existing with an evident lesion

in the central nervous system frequently is overlooked in the absence of urinary symptoms. It is peculiarly true, however, that in the presence of a lesion in the central nervous system, retention is more often present over a long period of time without causing evidence of renal insufficiency or urinary disturbance than it is in the presence of prostatic obstruction. As a result of dysfunction of the nerves supplying the bladder, the cystoscopic appearance is pathognomonic and gives a picture of the so-called cord bladder. Rectal examination in cases of this condition is often of diagnostic value. Instead of the normal prostatic fullness, a definite depression or flaccid condition of the rectal musculature in the prostatic region may often be discovered. On introduction of the finger, attention may be called to the possibility of disturbance in innervation by definite weakness of the anal sphincter and lack of muscular tonus in the rectal wall.

Another lesion which may cause considerable urinary retention is diverticulum of the bladder. Frequently the diverticula are multiple and may be even larger than the bladder itself, so that the resulting retention is of marked degree. Since the urine in cases of diverticula usually becomes markedly infected, the presence of diverticula should be suspected in every case of urinary retention with marked pyuria. Although there is usually more or less pyuria and dysuria in cases of diverticula, occasionally there are no symptoms referable to the urinary tract and on rectal examination there may be little, if any, prostatic enlargement. Diverticulum nearly always occurs secondary to some from of obstruction at the vesical neck, usually with enlargement of the prostate gland. Its diagnosis can be made either by cystoscopic examination or, better, by means of a cystogram which visualizes the diverticulum as a rounded area of sacculation extending laterally from the outline of the bladder. After the bladder is emptied, a subsequent roentgenogram usually will show that a considerable amount of the opaque medium has remained in the diverticulum.

A method which should be employed more frequently in general diagnosis, and which is easily available wherever there is roentgenographic apparatus, is cystography. The technic of making a cystogram is comparatively simple, and consists of emptying the bladder and then injecting several ounces of a solution of 5 per cent sodium iodide. Exposures are then made, from a right and from a left angle; the bladder is emptied, and a third roentgenogram is made. Not alone will diverticula be readily outlined by this simple method, but other deformity of the bladder, such as the elevation of the base that occurs with prostatic hypertrophy, sacculation, or deformity of the bladder, and filling defects which occur with intravesical or extravesical tumor may also be demonstrated. The method is a routine in every case of suspected urinary retention in the clinic.

In regard to the treatment of advanced urinary obstruction, it should be emphasized that when the bladder has been discovered to be greatly distended, it should not be emptied immediately. In fact, as soon as it is evident that a large amount of urine is being retained, it would be best to hospitalize the patient and gradually to empty the bladder. One of the favorite methods is by means of inserting an indwelling urethral catheter and elevating the receptacle into which the urine is emptied so that the bladder is gradually drained by the process called decompression. The bladder should not be completely drained in less than twenty-four hours; preferably as long as forty-eight hours should be allowed. It has recently been suggested that decompression can be accomplished equally well by gradually draining the bladder through a ureteral catheter of number 5 or 6 caliber. The treatment directed against secondary renal insufficiency consists largely of forcing fluids, both by mouth and intravenously, a diet low in protein, hot packs daily, and general care preliminary to removal of the obstruction.

AN ANOMALOUS EXTRA-VESICAL ORIGIN OF URETER*

Report of Case

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Saint Paul

THE case to be reported is of unusual interest because of its bizarre nature, the great rarity of the condition presented, and the unique surgical problem peculiar to it.

A young woman, aged 25, was referred for urologic examination July 31, 1929. She complained of continual persistent drainage of urine from the region of the vulva. The exact origin of the drainage, whether from the urethra, vestibule or vagina, she did not know. The seepage of fluid had been persistently present since birth. Its rapidity was not influenced by coughing, straining or change of position. It was continuous day and night. It appeared, therefore, to lack the characteristics of urethral incontinence. Careful inquiry elicited no history of injury or surgical procedure to account for a traumatic vesico-vaginal fistula. Menstruation was normal.

These facts of history suggested the possibility of one of the rare anomalies of extra-vesical origin of a ureter (improperly referred to as termination of a ureter). With this in mind the detailed physical examination was designed to discover such an anomalous ureteral opening if present in the external genital region, or vagina, or to determine the cervix as the source of drainage, in case of origin in the uterus or a fallopian tube.

The patient was well developed and nourished. The temperature, pulse rate and respiration were normal. She did not appear ill. The general physical examination was quite negative. Only details pertinent to the condition complained of will be given.

There was no tenderness to pressure in either flank and no reaction to Murphy percussion. Neither kidney was palpable. No tenderness and no mass was discovered in the abdomen. The reflex reactions were present, normally active and equal on the two sides. Skin sensation was grossly normal and there were no areas of anesthesia or paresthesia.

Urinary tract radiographs showed kidney shadows of essentially normal size, configuration and position and no abnormality in other respects.

Urination was normal, the stream being of good size and force. There was no residual urine.

The skin about the genital region and inner aspects of the thighs was moist and slightly reddened. Careful inspection of the vulva and introitus disclosed no abnormality except wetness of the surfaces. The source of the moisture was not evident. The external urethral meatus was in normal position and of normal appearance. The urethra felt quite normal to palpation against the pubic arch. Para-urethral gland duct openings were present but each would admit a fine probe only a few millimeters. The vestibule and margins of the introitus were carefully searched with a fine probe but no abnormal opening could be found.

On bimanual pelvic examination a normal cervix, a freely movable uterine fundus in normal position, and normal adnexal regions were found. In the right lateral vaginal wall extending downward from the fornix was a flap-like ridge of vaginal wall. This structure became less prominent toward its lower end and disappeared altogether two or three centimeters from the introitus. With the patient in the knee chest position the vagina filled well with air permitting careful and complete examination of its whole interior. The cervix appeared normal. It was wiped dry and watched for several minutes. No fluid appeared at the os. The vaginal surfaces were searched with a probe, particularly the flap-like ridge on the right wall, but no opening was discovered.

On urethro-cystoscopic examination no anomalous ureteral meatus could be found in the urethra. The bladder neck and sphincter margin were normal. The bladder mucosa and contour were normal throughout. Both ureteral orifices were present, in normal position and were normal in appearance. Indigo-carmine, given intravenously, appeared promptly and in good concentration in each ureteral jet.

It was determined to let this temporarily conclude the examination.

*Case Presentation before the Minnesota Academy of Medicine, St. Paul, Dec. 11, 1929.

Upon removal of the cystoscope the vestibule was sponged dry and re-inspected. Suddenly the mucosa just to the right of the urethral meatus became wet. Grossly, no opening indicating the source of the fluid was evident. However, a fine urethral catheter, used to probe the region, finally slipped into a minute opening. It was situated on the vestibule one-half centimeter to the right of the urethral meatus. The catheter was passed into the opening twenty centimeters. A radiograph made later showed the catheter to extend upward parallel to the position of the vagina. In the region of the bladder the catheter was coiled up in a space extending upward and to the right. The catheter could now be felt beneath the flap-like ridge in the right vaginal wall.

The cystoscope was re-inserted and a shadowgraph catheter was passed for the full length into the normal right ureteral orifice. The radiograph showed this catheter to occupy a normal position of a right ureter (Fig. 1).

The small catheter which had been introduced into the opening in the vestibule near the urethra was now injected with sodium iodide solution. The radiograph made following this (Fig. 2) showed it to outline very well a tract extending upward parallel to the vagina and a cavity in the region of the bladder in which the catheter was coiled up. Extending upward from this, the solution outlined a long narrow tract in the position of a right ureter and ended in the upper renal area in a bulb-like extension, characteristic of the high lying, rudimentary upper pelvis of a duplicated kidney. The catheter which had been passed into the normally placed right ureteral orifice within the bladder was now injected and another radiograph was made (Fig. 3). It showed this ureter well filled, of normal position and communicating with the lower pelvis of the duplicated kidney.

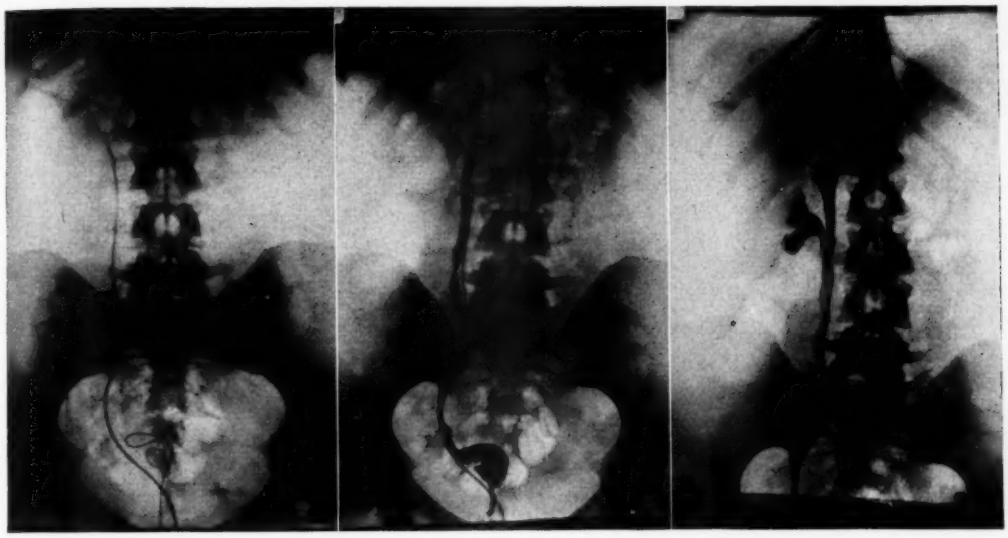

Fig. 1 Fig. 2 Fig. 3

The condition demonstrated was: Complete duplication of the right upper urinary tract with anomalous extra-vesical origin in the vestibule of the ureter draining the upper pelvis.

This condition represents an anomaly of the upper urinary tract—in fact, two anomalies: (1) the complete duplication of pelvis and ureter due to an anomalous cleavage of the ureteral bud; (2) the extra-vesical termination of one ureter in the vestibule due to an anomalous persistence of the attachment of the ureter to the Wolffian duct permitting the ureteral orifice to be carried down with the downward shifting duct, so that either it comes to have finally its own separate opening on the surface or remains attached to the vestigial remnant of the Wolffian duct, Gartner's duct, and reaches the surface through this channel.

Anomalies of the upper urinary tract have been classified as anomalies of form, of number and of position. Such classification seemed to me purely arbitrary and without foundation in em-

bryology. Some time ago I submitted a new classification based upon the facts of embryology concerning the morphogenesis of these malformations.

CLASSIFICATION OF UPPER URINARY TRACT ANOMALIES

- KIDNEY (*Renal Blastema*)
  - Position
    - Under-rotation
    - Over-rotation
    - Ectopia
    - Incident to Malformation
  - Form
    - Fusion
      - Horseshoe
      - Tandem
      - Lump
    - Infantile
    - Absent
    - Polycystic?
- URETER AND PELVIS (*Wolffian Duct*)
  - Cleavage
    - Partial Duplication
    - Complete Duplication
  - Origin
    - In Bladder
    - In Urethra
    - In Genital Tract
    - In Vestibule
    - In Rectum
  - Structure (Narrowing)
    - Calyx
    - Ureter
    - Uretero-Vesical Orifice
- BLOOD VESSELS
  - Division
  - Number

According to this classification anomalies of the upper urinary tract have been grouped as:

1. Anomalies of the secreting structure, the kidney proper, derived from the renal blastema.
2. Anomalies of the excreting structure, the ureter, pelvis, calyces and collecting tubules, derived from the Wolffian duct.
3. Anomalies of the renal vascularization.

The first group, anomalies of the kidney proper, result from faulty development in the renal blastema and from abnormality in the ascent and rotation of the kidney derived from this structure. The anomalies of form, such as horseshoe kidney, lump kidney and possibly polycystic kidney, and anomalies of position, such as ectopic kidney and under-rotated or over-rotated kidney of the old classification, fall into this group.

The second group, anomalies of the ureter, pelvis and calyces, result from faulty development in the ureteral bud arising from the Wolffian duct. The "anomalies of number" of the old classification, frequently spoken of as duplicated kidneys, fall into this group under the heading "cleavage anomalies." These are due to premature cleavage of a single ureteral bud or the formation of two buds.

Anomalous origin of the ureter was not taken into account under the old classification. Such anomalies are due to failure of the ureteral bud stalk to be dropped off in its normal definitive position in the bladder. Instead its attachment to the Wolffian duct persists abnormally long or permanently and permits it to shift downward with the Wolffian duct to open below the level of the bladder. This occurrence provides opportunity for origin of the ureter in the urethra in both sexes, in the common ejaculatory duct of the male or in Gartner's duct, or in the vestibule of the female. When it is combined with other departures from normal, it may give rise to origin of the ureter in the rectum of either sex, in the seminal vesicle or vas deferens in the male and to origin in the fallopian tube, uterus or vagina in the female.

Abnormal structure in the form of narrowings of the excreting channels developed from the ureteral bud gives rise to congenital strictures. They may affect the excreting channels anywhere from a calyx within the kidney to the ureteral opening in the bladder mucosa. They have been grouped under "structure."

The third group, anomalies of the renal blood vessels, occur as abnormal divisions and distributions of an otherwise normal renal artery or vein or as supernumerary vessels originating from aorta or cava or other neighboring vessels.

In the case reported, the cleavage anomaly, consisting in complete duplication of pelvis and ureter, obviously is due either to two ureteral buds having formed on the Wolffian duct or to the premature division of a single ureteral bud.

Under normal development, that portion of the Wolffian duct between its opening into the bladder or cloaca and the point of origin of the ureteral bud, spreads into the expanding bladder wall until the stalk of the ureteral bud is reached. The latter then shifts onto the bladder as a separate opening, its definitive position in the bladder being thus established. The expanding bladder

wall carries this ureteral opening lateralwards, and upwards relative to the now separate Wolffian duct opening in the bladder. In addition there is a medial, and downward shifting of the latter opening which further contributes to this relative position of the ureteral orifice and the continuing attachment of the Wolffian duct to the bladder.

Should there have been two ureteral buds, the stalk of the first one, the lower one, will first meet the bladder and shift onto it as the ureteral opening of the lower pelvis. The second stalk continuing its attachment to the Wolffian duct usually meets and shifts to the bladder before the duct attachment is below the position of the vesical neck. This stalk, representing the ureter of the upper pelvis, however, will have been carried by the Wolffian duct into a position more medial and lower than the opening of the ureter of the lower pelvis. Thus it is that the lower medial ureteral orifice in the bladder of a complete duplication, drains the upper pelvis, while the upper lateral orifice drains the lower pelvis.

In the case of a complete duplication arising from the premature cleavage of a single ureteral bud instead of from two buds, it must be presumed that the stalk of the ureteral bud either alone, after shifting to the bladder as a separate opening, or together with the Wolffian duct before this occurs, goes through this same process of spreading into the expanding bladder wall until the point of ureteral bifurcation meets the bladder wall, whereupon, in succession, first one and then the other division of the ureter will shift to the bladder as separate openings.

In the case reported, the anomaly of origin of the ureter, extravesically in the vestibule, is due to the upper ureteral bud stalk (if there were two ureteral buds) or the upper division of a bifurcated stalk (if there was premature cleavage of a single ureteral bud) failing to shift to the bladder as a separate opening, as did the ureter of the lower pelvis. Instead its attachment to the Wolffian duct either persists abnormally long or is permanent. In the first case it would represent a downward carriage by the Wolffian duct of maximal extent to the vestibule, finally shifting to this site as a separate opening. In the second case it would represent a permanent attachment of ureter to Wolffian duct which reaches the surface via the vestigial remnant of this duct as a patent Gartner's duct.

The case reported presents a unique surgical problem peculiar to such conditions. In cleavage anomalies of the upper urinary tract there may be no symptoms and no associated pathologic change. Under these circumstances there is no indication for treatment, surgical or otherwise.

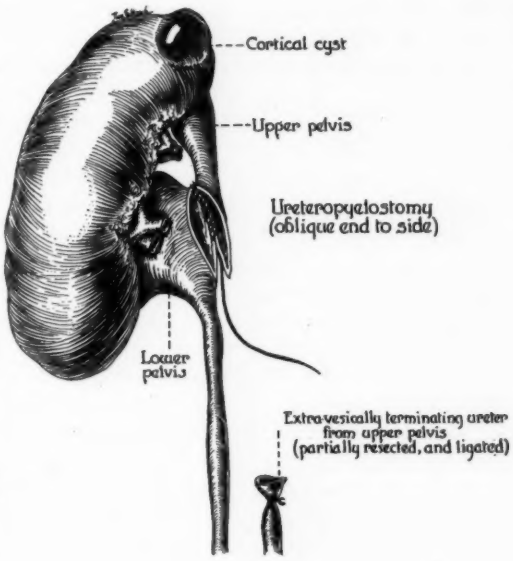

Fig. 4

Frequently, however, the anomaly is present in association with an obstructive lesion and there may be dilatation, persisting infection or stone formation, or tuberculosis or neoplasm may be present. Under these circumstances the problem of surgical treatment is rendered unique and peculiar because of the associated anomaly. In general, the major surgical procedures which come up for consideration are, nephrectomy, hemi-nephrectomy and finally the possibility of anastomosis between the two divisions of the double kidney.

Anastomosis between the two divisions is useful as applied to obstructive lesions affecting one division of a duplicated upper urinary tract, the anastomosis providing drainage for the obstructed division through the ureter of the unobstructed division.

By hemi-nephrectomy it may be possible to remove the diseased half of the double kidney, thus preserving the function of the undiseased half. In my own experience, hemi-nephrectomy has been employed four times, in all cases with

an excellent functional result. In a fifth case, hemi-nephrectomy was undertaken but because of the particular vascular arrangement and the extensive changes of a suppurative calcareous pyonephrosis involving the lower division of the kidney, the procedure had to be abandoned in favor of complete nephrectomy. The experience with hemi-nephrectomy which has been mentioned is a particularly fortunate one and such results with the procedure cannot be regularly expected. It has many undesirable features, among which may be mentioned great technical difficulty because of the particular vascular arrangement, the danger of immediate or secondary hemorrhage from the sutured cut surface of the remaining portion of kidney and urinary fistula or chronic recurring accumulations in the wound due to remaining portions of the parenchyma which had drained into the removed pelvis.

Anastomosis between the two divisions of the duplicated upper urinary tract avoids the dangers of hemi-nephrectomy, and when conditions are suitable for its employment it is a much more desirable procedure than hemi-nephrectomy. My first operation of this sort was employed more or less as a makeshift means out of a technical difficulty. The result was excellent. My first impression that this was the first operation of the kind to have been employed was incorrect. A year or so following this first operation the same procedure was carried out in a second case with an equally satisfactory result.

In the case reported, relief of the persistent extra-vesical drainage of urine might be accomplished in various ways: total nephrectomy, hemi-nephrectomy, simple ligation of the anomalous ureter, transplantation of the anomalous ureter into the bladder, or by uretero-ureterostomy or uretero-pyelostomy between the two divisions of the duplicated upper urinary tract.

Certainly in this case the lower half of the kidney is well worth saving and conservatism contra-indicates total nephrectomy. Although the upper division is of very little functional importance its removal by hemi-nephrectomy is not indicated because of any pathologic change apart from the extra-vesical opening of its ureter. This undesirable functional procedure should not be undertaken because of the extra-vesical drainage, if this can be corrected by a safer procedure assuring an equally satisfactory functional result. Simple ligation of a ureter without removal of the secreting parenchyma which it drains may be employed as has been done when the ureter is inadvertently cut during hysterectomy. If the kidney is uninfected and complete atrophy promptly results from the complete obstruction of ligation, a favorable result may be expected. On the other hand, particularly in the presence of infection, serious difficulties may ensue. Transplantation of the ureter into the bladder is a technically difficult procedure and does not regularly give a good functional result, devoid of possible complications. The exposure is difficult and transplanting the ureter so as to provide a competent uretero-vesical valve is not easy. A uretero-vesical anastomosis which does not provide against intra-vesical pressure being conducted into the ureter assures damage to the kidney above and provides an excellent probability for ascending infection and complications of real consequence.

The above considerations indicate transplantation of the anomalous ureter into the lower pelvis or ureter as the operation of choice in the case reported. Accordingly uretero-pyelostomy as illustrated (Fig. 4) was performed. The segment of ureter between the point of anastomosis and the brim of the bony pelvis was resected.

The patient made an uneventful convalescence and now more than three months since operation she remains entirely relieved of her distressing symptoms without any pain referable to this upper urinary tract or urinary disturbance of any sort and in all respects is quite well.

CORONARY OCCLUSION WITH SURVIVAL*

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CORONARY occlusion is most often a dramatic cause of fatality, but that many cases do survive is becoming more universally recognized as the clinical picture is becoming more familiar. Herrick[1] recognized this fact as far back as 1919 when he classified coronary thrombosis cases as follows:

1. Cases of instantaneous death in which there is no death struggle.
2. Cases of death within a few minutes or hours after obstruction.
3. Cases of severity in which death is delayed for several hours, days or months, or recovery occurs.
4. A group that may be assumed to exist embracing cases with mild symptoms; for example, a slight precordial pain ordinarily not recognized as due to obstruction in the smallest branches of the coronary arteries.

Only three articles, in the enormous amount of literature which has recently sprung up on coronary occlusion, have been devoted entirely to the consideration of these milder cases with recovery.

Luten[2] reported three cases in 1927 which survived one year five months, one year six months; and one year, ten months, respectively.

Levine,[3] in 1925, reported five cases with survival to the time of writing of from eight months to one year ten months.

Pardee[4] reported one case living up to time of publication in 1927, three years and three months.

Hamburger,[5] in an article not devoted exclusively to cases with recovery, reports he has seen several cases that have led fairly active lives for a period of five years or longer, giving no specific figures.

Willius[6] makes a statement that one-half the cases of coronary occlusion die immediately, and one-half survive a few days to months, or even years.

The reason for survival lies in the fact that only a small vessel may become occluded and, therefore, only a small area of heart muscle be infarcted; or, granted that a large area be infarcted, the capillary anastomoses are capable of establishing a collateral circulation to maintain nourishment to prevent a fatal thinning, scarring and final rupture of the ventricular wall. Indeed, such a rupture is sometimes the late result of an old infarction from which the patient may have survived a considerable length of time.

Wearn[7] has demonstrated beautifully the capillary bed of the heart and anastomoses formerly thought to be non-existent in the coronary circulation (Fig. 1).

A brief review of the clinical picture of coronary occlusion is presented below:

SYMPTOMS OF CORONARY OCCLUSION

Subjective

Often history of preceding attacks of true angina pectoris with normal electrocardiogram and no nitrite relief.

Terrible agonizing pain in precordium radiating down left arm.

No relief from nitrites or morphine.

Fear of impending death.

Vise-like choking feeling.

Objective

Age over 55.

Patient in shock.

Cold, clammy forehead and extremities.

Irregular rapid pulse.

Change in heart tones.

Murmurs, later pericardial friction rub.

Fever.

Slight leukocytosis.

Low blood pressure.

Pulmonary signs.

Changes in electrocardiogram.

The electrocardiograph affords the best confirmatory evidence when the so-called Pardee's phenomenon[8] is present. This change occurred in seven of our ten cases. Other suggestive changes, especially if known not to exist before, were: auricular fibrillation (1

*Presented before the Southern Minnesota Medical Association, Winona, Minnesota, June 15, 1929.

case), bundle branch block (2 cases), slurring or notching of QRS complexes (1 case).

Pardee has called attention to a fairly characteristic electrocardiographic sign illustrated in some of these curves. The S-T takes off the R-wave in one or perhaps two of the leads above or below the iso-electric point and is convex upward to curve downward into a negative T-wave. When found, this sign is very suggestive but does not occur in all cases. Wearn[9] concludes: "It is obvious there is no one form of the electrocardiogram characteristic of this condition." Only one out of ten cases reported by him had Pardee's typical sign. Clark and Smith[10] state: "No alteration of the T-waves in coronary thrombosis has been accepted by the many writers as characteristic. For clinical purposes this change consisting of elevation of the T-wave which branches from the R or S limb may be considered as strong presumptive evidence of coronary occlusion." Fred M. Smith[11] showed the electrocardiogram of a dog with typical inversion of T1 and T2 with convexity of the S-T interval following ligation of the coronary arteries.

Our case reports call attention again to the frequency of coronary disease and angina pectoris in physicians—three in our small series of ten cases. Survival in our longest case so far has been five years and two months, and this patient subsequently died of cerebral hemorrhage. Seven of the cases have so far survived more than two years, and nine of the ten cases more than one year.

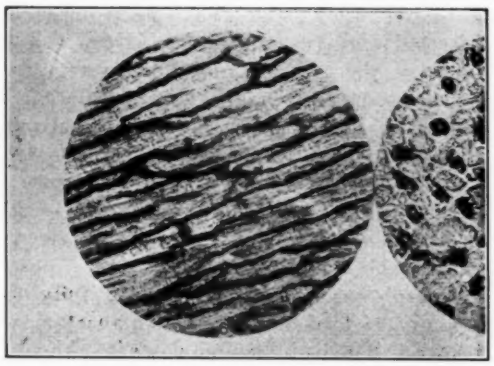

Fig. 1 (Wearn)

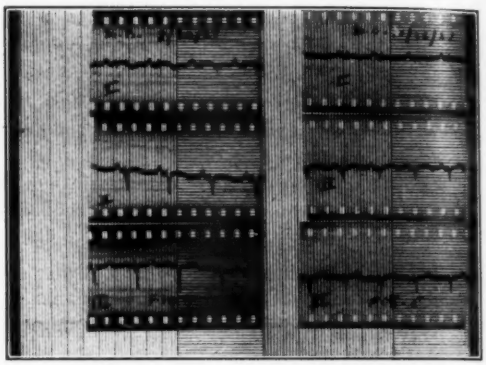

Fig. 4 Fig. 5

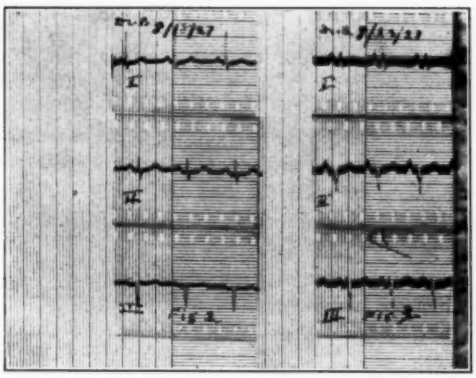

Fig. 2 Fig. 3

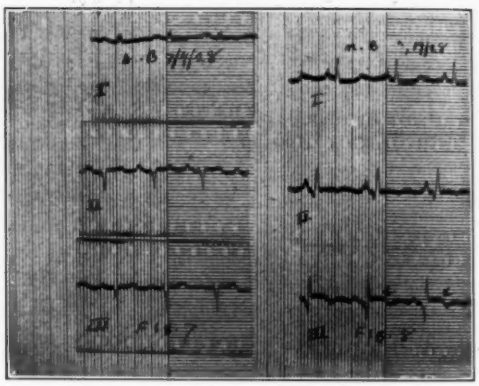

Fig. 6 Fig. 7

CASE REPORTS

Case 1.—Dr. B., male, aged 69, experienced a mild precordial pain while hunting. His electrocardiographic tracing was normal (Fig. 2) the morning of the same day infarction occurred. That night, and for the next week, he presented all the classical signs of coronary infarction enumerated earlier in this article. Four days after infarction TI had become flat (Fig. 3), but

not until twelve days after occlusion did Pardee's phenomenon occur (Fig. 4).

Note in Figure 4 that S-TI is above the iso-electric line and TI has become negative with the S-T interval convex upward. Two months later the patient had resumed normal activity. The tracing then showed (Fig 5) improvement in an upright T2. A year after infarction (Fig. 6), Pardee's phenomenon has disappeared, but TI is still flat. He has no more pain but a tendency toward congestive heart failure when too active.

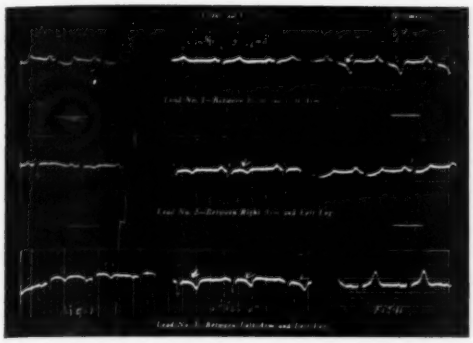

Fig. 8

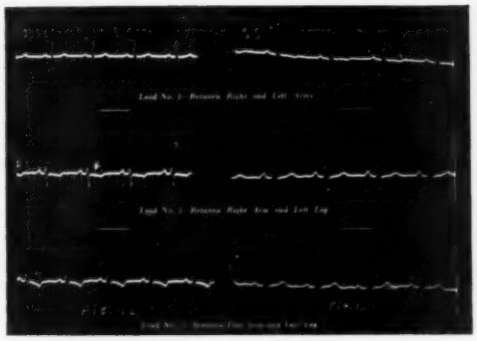

Fig. 9

Fig. 10

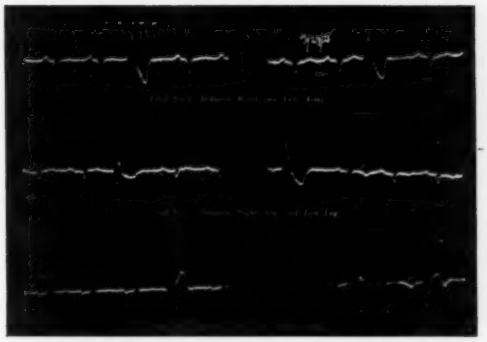

Fig. 13

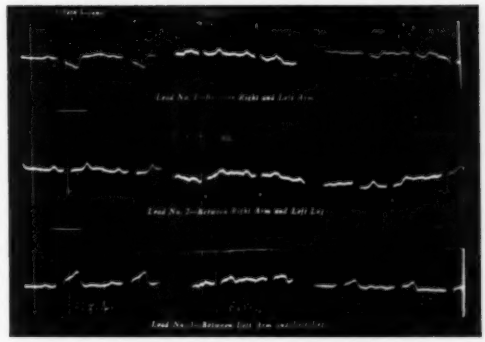

Fig. 14

Fig. 11

Fig. 12

Fig. 15

Fig. 16

Fig. 17

Case 2.—Angina is notably absent in congestive failure as shown by our second case, a woman of sixty-four, who died two years after her occlusion with pulmonary edema and general anasarca. She had repeated attacks of angina till congestive failure set in, with Pardee's sign in the electrocardiogram (Fig. 7). After that, she was free from pain till compensation was restored, when attacks of precordial pain recurred. Subsequently, a second and final period of decompensation brought relief from pain.

Hamburger[5] indeed reported three patients who suffered but very little pain but died of congestive myocardial failure based on coronary occlusion. In these cases the occlusion may be very gradual.

Case 3.—L. M., a female aged 62, had had gradually increasing dyspnea for two weeks previous to falling in a faint two days before the electrocardiogram was taken. No history of cardiac pain could be elicited. Physically she had marked cardiac hypertrophy, a palpable liver edge, sacral edema, orthopnea, cyanosis and blood pressure of 86 systolic and 54 diastolic. The white count was 9,600, the temperature 102. She also had a stone in her left kidney pelvis and pyuria. She was discharged five weeks later in a good state of compensation and, as far as we know, is in fair health three and a half months later. She illustrates a painless type of infarction in whom the diagnosis could only be guessed without electrocardiographic confirmation demonstrating Pardee's phenomenon (Fig. 8).

Case 4.—John M., a male aged 59, suffered from very severe precordial pain for five days with fever, rapid pulse and collapse. A precordial friction rub developed. The electrocardiogram was taken one year after infarction and still showed Pardee's phenomenon in the second and third leads (Fig. 9). He is in good health two years and five months later.

Case 5.—Dr. D., a male aged 62, dated his limitation of activities, due to precordial distress, to a single severe protracted attack of angina pectoris two years before this tracing was taken. Lead I shows Pardee's phenomenon (Fig. 10). His survival so far has been two years nine months.

Case 6.—F. M., a male aged 67, had his first attack of

angina pectoris four and one-half years ago. In February and July of 1924 his electrocardiogram showed a negative TI at another clinic. In January and July, 1925, his tracings were reported practically normal. After freedom from symptoms for three years a Pardee's sign was present in his electrocardiogram four days after a severe second attack (Fig. 11). Two and a half months later there was only a negative TI and Pardee's sign has disappeared (Fig. 12). He is alive and working, free from further attacks four and one-half years after his first infarction and one and three-fourths years after his second attack.

Case 7.—W. M., a male aged 54, suffered a very severe attack of precordial pain accompanied by an irregular pulse, temperature of 99.8, and had a white cell count of 14,150. He had had an old luetic infection which had been treated and now has a negative Wassermann. Two days after his infarction he had a flat TI and numerous extrasystoles (Fig. 13). Three months later the TI was again positive but Pardee's

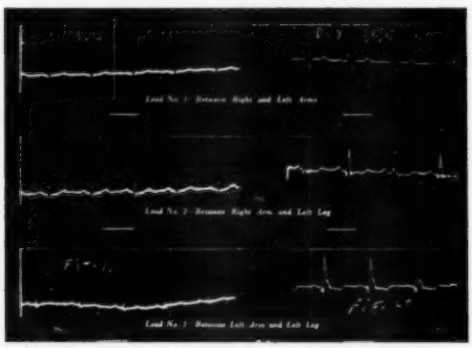

Fig. 18 Fig. 19

phenomenon was present in Leads II and III (Fig. 14). He is an active insurance agent one and one-half years later.

Case 8.—J. W. G., a male aged 68, had had repeated typical attacks of angina pectoris for the past eight years before his first electrocardiogram was taken, which showed a right bundle block (Fig. 15). Six months later, during which interval he was free from attacks, a second tracing showed the bundle branch block had disappeared (Fig. 16). A third tracing four and one-half months later, about two months after a severe attack of angina, then showed the bundle branch block had reappeared and, in addition, an auricular fibrillation (Fig. 17). This patient also had gallstones and a diverticulum of the esophagus. He is still living a fairly active life one year after his last infarction, free of angina with good compensation under digitalis.

Case 9.—Joe M., aged 64, had an infarction two and one-half years ago, accompanied by severe precordial pain, vomiting and hemoptysis. The attack was followed by left basal pulmonary infarction, and, later, signs of embolism in the left posterior tibial artery and fresh pulmonary infarctions. The diagnosis was infarction of interventricular septum due to occlusion of the descending branch of the left coronary artery with mural thrombus on both sides of the septum, casting off emboli to both the pulmonary and systemic circulations. The tracing was taken one month after infarction and showed no abnormality of the T-waves, and only a slurring and notching of the QRS complexes in Leads II and III with low voltage (Fig. 18). This man's activities are limited by dyspnea and precordial pain, two and one-half years after infarction.

Case 10.—Dr. Y., male, aged 66. Five years before this patient's tracing was taken he had an attack of angina pectoris diagnosed coronary infarction. He led a very active country practitioner's life ever since without any distress. At the date of this curve, he was suffering from thrombo-angiitis obliterans. There were also signs of arteriosclerotic kidneys, marked peripheral arteriosclerosis, and two months after this tracing he died of cerebral hemorrhage. This case illustrates the effects of universal vascular disease. The electrocardiogram shows a left bundle branch block (Fig. 19). He survived his coronary occlusion five years and two months to die other than a cardiac death.

CONCLUSIONS

1. Ten cases of coronary occlusion are reported with survival—the longest duration being five years two months. Two of these cases survived at least two occlusions besides having attacks of angina pectoris.

2. The electrocardiogram offers substantial corroborative evidence of coronary occlusion, and, of the changes found, Pardee's sign is the most suggestive (seven out of ten cases).

3. Pardee's sign was found to appear as early as the second day and to still be present, in some cases, as long as two years after occlusion took place.

4. Many cases survive minor occlusions, and rare cases extensive infarcts.

5. Death may occur in these cases months later from congestive myocardial failure with or without further attacks of precordial pain.

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DISCUSSION

Dr. C. N. Hensel (St. Paul): The problem of coronary infarction and its relation to the subsequent viability of the patient has been beautifully presented to you today. It is an interesting fact that these cases of proved infarction can survive such an insult to the heart provided the condition is recognized at the onset and the treatment guided by both clinical and electrocardiographic findings. This characteristic cresentic shaped deflection of the T-wave is the best diagnostic criterium and the cases that recover may show a return to the normal contour.

This problem of coronary infarction is not only of interest to the internist but also to the surgeon. Frequently in cases of coronary infarction, the pain originates in the abdomen, only secondarily rising to the lower sternum, and may cause symptoms simulating acute abdominal disease such as gallstone colic. Cardiograms in these cases may show the characteristic changes in the T-wave and thus forewarn of grave cardiac condition and perhaps cause postponement of surgical intervention.

I recall one such case of a woman of seventy years of age who had had repeated attacks of gallstone colic for many years. The present attack was of undue severity and attended with more than the usual prostration. She was taken to the hospital and watched for twelve hours under the exclusive care of a very competent surgeon. There was tenderness over the gallbladder region, muscular rigidity and pain over the lower part of the sternum. Slight fever and leukocytosis were found. At operation a gallbladder full of stones was found but because of her poor general condition and of a rapid pulse the operation was quickly terminated, the gallbladder being left in place and drainage instituted. When seen at six o'clock that evening by her attending physician her condition seemed to be satisfactory. At twelve she awakened from a drowsy sleep gave a few gasps and was dead. At autopsy the pericardial cavity was found filled with blood. When the heart was removed one could detect an area of softening in the wall of the left ventricle in which there was a small tear entirely through the wall. It was from this torn area that the hemorrhage took place, which caused her death. The microscope revealed that this area of softening was due to a previous coronary infarction and undoubtedly this infarction was responsible for her pain simulating her gallbladder attacks. It seemed probable that the strain of the operation and the subsequent nausea had been sufficient to rupture the heart. Had an electrocardiogram been made previous to operation it might have warned the surgeon of the serious damage to the heart and the fatal issue might have thus been avoided.

Since we have been using the electrocardiograph more routinely we have come to recognize that there are patients who have the angina pectoris syndrome without coronary infarction, but who eventually develop infarction of tiny branches of the coronary arteries, accounting for the more prolonged attacks of anginal pain. In these cases usually the final major attack of pain will cause the death of the patient and at autopsy multiple areas of fibrosis in the heart muscle are found, indicating previous infarctions. So the electrocardiogram is frequently of value in differentiating simple angina pectoris from mild coronary infarction.

I have a case to report with a few lantern slides which illustrates Pardee's phenomenon in the T-wave that Dr. Evans has shown through his series. The patient, a Jewess, fifty-six years of age, a diabetic for five years, was seized suddenly after a hearty evening meal with intense pain in the middle of the sternum. My associate was called and found a bounding pulse and strong heart tones. Blood pressure was 220 systolic and 110 diastolic. Surrounded by anxious relatives and greatly excited herself, it was difficult to be sure that the patient was really ill. Nitrites and morphine brought some drop in blood pressure and some relief of the acute pain, but a dull sternal pain persisted for several days. An electrocardiogram the following day showed a slight coronary T-wave in Lead 1 with a deeper drop on the second day, subsequent records showing a gradually rising T-wave till at the end of three weeks the T-wave had lost its coronary curve and was only slightly flattened in Lead I.

The patient has now lived a year and a half following this attack of coronary infarction. Of course we know that diabetic patients have as a rule a generalized arteriosclerosis and are susceptible to coronary infarction. This case illustrates nicely what happens with recovery. I have had experiences with other cases illustrating the same points Dr. Evans has shown. Some of them make clinical recoveries, others do not die, but they drag around with a persistence of marked debility and greatly limited heart capacity. The cases that recover their former energy show eventually a return of the T-wave to the upright position. The cases that do not recover their customary energy usually show on their cardiographic records a persistence of the downward declination of the T-wave or a marked flattening of the T-wave, indicating grave myocardial damage.

The cardiograph is becoming of increased value as an aid to differential diagnosis, and as a guide to treatment.

Dr. George Eusterman (Rochester): I want to bring out two points. The other day I had a patient

who had been with us seven years previously and we had made a diagnosis of generalized arteriosclerosis. He came back presenting gastro-intestinal disturbance, largely gastric. No evidence of disease of the gastro-intestinal tract or biliary tract was found. There was a little history of dyspnea. The heart was of normal size and normal rhythm. There was evidence of some torsion of arch of the aorta on the film. Generalized arteriosclerosis was present as before. The blood pressure was normal. The patient was reassured that there was no organic gastric disease. Two weeks later the brother wrote that after a hearty dinner the patient went out and played golf, and died suddenly on the course.

Willius says that practically 50 per cent of those with infarction of the coronery arteries die in the first attack.

One other point is the sensitivity of the patient with angina pectoris and coronary infarction. Libman brought this out at the meeting of the Association of American Physicians. Dr. Evans brought out the point of the degree of pain. Libman feels that patients vary in sensitivity to pain and that insensitive patients may have definite infarction without any pain; he demonstrated this point in three or four patients in the wards at Mt. Sinai, using pressure on the styloid process as the index. Crohn feels that patients whose first symptoms of gastric disease are hemorrhage or perforation or obstruction are insensitive persons who do not have the usual symptoms. The same is true of patients with cancer who go on to inoperability before symptoms occur. I want to congratulate both Dr. Evans on his presentation and Dr. Hensel on his discussion.

JAUNDICE PROBLEMS*

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JAUNDICE, the symptom, is forever flashing itself before us. Sometimes it is thought of as something likely to appear during the course of a disease under observation and then it does not surprise us when it comes, but more often it appears unexpectedly as a seemingly isolated sign. It always rouses curiosity and is always a puzzle. Wonder as to its cause, query as to its treatment rise up like a blank wall. There is a feeling akin to helplessness. I suppose almost every doctor has had that experience.

Of course the various etiological factors pass before one's mental vision, but, even so, diagnosis is not always so easy. Besides, there are cases that do not permit of a thorough clinico-scientific study. The patient may refuse special methods of examination, may be unfit to bear such methods, may be remote from a place where such technical work is available and may refuse to be moved; also, the patient's history may be difficult or impossible to secure. All these and perhaps other factors make it necessary for the doctor to know the clinical picture of the various conditions presenting the common symptom of jaundice and to be able to thread his way through the diagnostic labyrinth with a minimum of special technical methods. An aged person presents jaundice as a salient symptom and the most careful analysis of recognizable concomitant symptoms may fail to enable us to say whether there be duct obstruction due to pressure from outside, changes within the walls of the duct, obstruction from within the lumen or changes in the liver substance or perhaps a jaundice of hemic origin. If surgery or autopsy be not done etiological diagnosis may never be made.

A few actual cases will be discussed, as that is the best way to draw parallels and contrasts.

Case 1.—A woman thirty-eight years of age and the mother of several children said she had been quite well up to two months prior to admission to the hospital. She then began to feel tired and indisposed, lost her appetite, felt nauseated much of the time and occasionally vomited. She presents a Grade 1 icterus and admits upon close questioning that she first noticed this some three months ago, characterized by periodicity, a gnawing pain in the stomach at a variable time after meals and relieved by food. She has never had an acute attack of pain. Continuing to question searchingly it is learned that she has had much indigestion over a period of many years and also alternating constipation and diarrhea. She states that her indigestion is better than it was, that it has varied in degree and that her stools have been and are a pale yellow. Her physician says the urine contains bile.

What is the etiological factor in this case? So-called acute catarrhal jaundice must be thought of but the variability of her icterus, its long duration, absence of fever together with her gastro-intestinal symptoms are certainly suggestive of chronic cholecystitis and secondary irritation of the colon. Perhaps a stone shifting its position between the cystic and common ducts would account for the variable jaundice. The patient was too ill to be subjected to a complete gastro-intestinal and Cole-Graham examination at the time. However, the Van den Bergh tests and blood count were done. The results both complicated and clarified the diagnosis. They were as follows:

Icterus index, 20.

Van den Bergh (direct) negative.

Van den Bergh (indirect) immediate and positive.

Blood picture: that of pernicious anemia.

Treated with liver extract her improvement was prompt and rapid.

The blood picture and indirect Van den Bergh would indicate icterus of hemolytic origin and made a different interpretation of the gastro-intestinal symptoms possible. But, why the pale stools? Certainly we do not expect such in pernicious anemia. May not cholecystitis also be present? At a later date when the patient's condition permitted, the Cole-Graham examination was made. Dr. T. G. Clement, roentgenologist, submitted the following report:

"G. B. not filled in 12 and 15 hours but well filled in 24 hours.

"Small dense shadow cystic duct suggesting stone. Delay in filling and emptying time of gall-bladder suggests and confirms cystic duct ob-

*Presented at the annual meeting of the Northern Minnesota Medical Association, Bemidji, Minn., Sept. 2, 1929.

struction." Here then is a case for multiple diagnosis—clinically the gallbladder symptoms masked those of pernicious anemia. The latter was not accompanied by nerve symptoms in this case. The positive Van den Bergh indirect test pointed away from duct obstruction and when supplemented by the blood examination the source of the jaundice became clear. It was of hemolytic origin. The long years of gastro-intestinal symptoms surely antedated the onset of the pernicious anemia. They were characteristic of gallbladder disease. The *x*-ray examination tends to confirm that diagnosis.

Case 2.—A white female, aged 25, married, was taken ill with headache, weakness, vertigo, nausea and vomiting. Then came labor pains, convulsions and coma. Labor was precipitate. She was then taken to the hospital, two days after the beginning of her symptoms. She was still unconscious and icterus was noticed for the first time. It was about Grade III. The record shows her to be poorly developed and poorly nourished, to have a badly neglected mouth, a normal blood pressure, impalpable spleen and liver, no edema. Other physical findings were essentially normal. The vomitus had been clear and watery at first but was coffee ground type when admitted to hospital March 15. It continued so for the next two days, by which time she had regained consciousness, felt languid and nauseated, but otherwise quite comfortable. Shortly the icterus was complicated with fever.

It may be assumed that a toxemia of pregnancy had existed and was subsiding, but what about the jaundice? Again the puzzle. Was it acute catarrhal jaundice or acute yellow atrophy? The languor, continued mental dullness and fever were not distinctive. They could be attributed to the recent eclamptic state. The fever could be attributed to a poorly contracted and poorly involuting uterus. It could be associated with an acute cholangitis. It could be accounted for by an acute hepatic degeneration. The latter is common enough in pregnancy to not excite surprise when it occurs. Admittedly it was too early to arrive at a definite diagnosis. Laboratory methods might help, but in their absence, and conceivably with them, time alone could solve the problem.

On the first, second and third days after admission her icterus index was 85, 89, and 187 per cent. On March 21, 8 days after admission, the Van den Bergh direct was four plus and the indirect was the same. The test for urobilin in the urine was negative. The ammonia nitrogen urinary output on March 19, 20, and 21 was 2.2 gm., 1.3 gm. and 0.5 gm., respectively. The blood examination on March 16 (next day after admission) was 78 per cent hemoglobin, 4,620,000 erythrocytes and 22,200 white cells. The next day the white cell count was 27,400.

Now, how may we interpret the laboratory reports to aid us in reaching a rational conclusion? It is true that acute yellow atrophy may be accompanied by headache, coma, and convulsions, but these do not precede the icterus, they follow it, and appear late in the affection. Thus we may be sure that in this case they were probably due to a pregnancy toxemia and that whatever may have happened to the liver in the way of acute degeneration, even though due to the same primary etiology, was of later development.

The laboratory findings of a high and rising white cell count point clearly enough to infection, sufficient perhaps to start degenerative hepatic changes. But a high leukocytosis is not in itself distinctive of acute yellow atrophy. The rising icterus index is common to all forms of obstructive jaundice and taken alone is not etiologically distinctive. The Van den Bergh tests, direct and indirect, are high. Again we find nothing definitely diagnostic. We may infer from them that all the bile secreted by the liver does not reach the intestine and that some change is probably going on in the liver cells, the nature of which we do not know. It might also be inferred that some of the jaundice might be due to blood destruction. However, the near normal erythrocyte count, 4,620,000, does not justify such a conclusion. The negative urinary urobilin findings also point away from hemolytic jaundice. If bile reaches the intestine it is in part converted into urobilinogen and urobilin. As such it is reabsorbed, taken to the liver and converted into bilirubin. If the liver is so badly degenerated that it cannot change urobilin into bilirubin it is probably also incapable of secreting bile; therefore urobilin would not be expected to appear in the urine in acute yellow atrophy. As the net result is the same in complete obstructive jaundice the failure to find urobilinuria does not help us much. The record fails to show the color of the feces. If an observation had been recorded indicating the presence or absence of bile in the stool, then this, taken together with icterus, would afford something tangible in the direction of an etiological diagnosis. We are denied this small help.

One function of the liver is deamidization. The laboratory record shows a marked increase in the ammonia nitrogen excreted in the urine. This is suggestive of failing liver function. As this patient had suffered a pregnancy toxemia we cannot be sure that an acidosis was not responsible for the high ammonia nitrogen excretion in the urine. The latter view is strengthened by the following rapid reduction in the ammonia

nitrogen output. Thus but little positive diagnostic aid is derived from the laboratory findings, which, by the way, are sometimes apt to be very confusing to one doing clinical work. As this patient began to improve very shortly, and was discharged from the hospital as cured at the end of two weeks, it is quite likely that the final diagnosis of acute catarrhal jaundice was correct. On the other hand we are not to assume that there was no degree of acute hepatic degeneration. The toxemias of pregnancy are known to be peculiarly causative of the syndrome mentioned. It may very well be that such a process actually started in this case but owing to the weakness of the toxemia, or its short duration, or to a high power of resistance by the patient, or all three influences together, resulted in recovery with but little damage. How little or how much damage may never be known. Perhaps the reparative scarring may be sufficient to cause symptoms of hepatic cirrhosis at some future time. Perhaps some day this patient may come to autopsy, when the pathologist will read the story of an old arrested acute yellow atrophy.

Case 3.—A male, aged 21, a laborer, sailor and world wanderer, was admitted to the hospital August 31 and died two days later. The record shows that his illness started about two weeks before entering the hospital. He felt weak, had headache and slight jaundice. A doctor sent him to bed for a week. He then worked two days and quit. There was progressive weakness and icterus, prostration, confusion, delirium and stupor —then death.

The past history and physical findings in this case are meager indeed. He was well nourished, had dental caries, icterus, and his liver and spleen were not palpable. These were the only notations of interest.

The laboratory report shows a normal blood count except for a slight leukocytosis of 10,000, an icterus index of 166, Van den Bergh negative direct and positive indirect. The diagnosis recorded is acute yellow atrophy.

Certainly this case record affords very little for purposes of scientific study. The course of the illness, however, contrasts markedly with that of the preceding case and confirms the statement that delirium, convulsions and stupor are late symptoms in acute yellow atrophy. Note that in this case the onset was gradual, the course progressive and the end fatal. Note that in the preceding case the onset was sudden, the icterus followed the convulsions, the progress was toward rapid recovery.

Now why should this apparently healthy young man develop this fatal malady following a seemingly normal condition? Certainly acute hepatic degeneration does not arise without cause. Certainly, also, dental caries as noted in the record could hardly be accepted as the probable cause. That this disease is due to an intoxication or infection of some sort there can be little doubt. Failing to find it in the clinical record we turn to the autopsy hoping for something informative. The autopsy report is submitted in brief:

Teeth are carious.

Liver weighed 1,000 gm.; is mottled with yellowish patches and softer than normal. Cut surface shows soft yellow patches alternating with purplish red patches. Bile ducts are patent. Acute cholecystitis present.

Small hemorrhages present in mesentery and kidneys.

Post-mortem diagnosis: Probably acute yellow atrophy, acute cholecystitis, dental caries, pulmonary edema, small hemorrhages into mesentary, omentum and kidneys.

On the basis of this report we can assume the mouth hygiene to have been extremely bad and accompanied with dental and gum infection. We could think of this as a possible etiological factor but acute cholecystitis should be considered in that role a great deal more logically. It could be considered more seriously if an associated cholangitis had been reported. The absence of any definite source of toxemia leaves us in the realm of speculation. One can't help but wonder if this homeless wanderer may not have been luetic, and, if so, had he been treated with antiarsenicals and been an individual sensitive to arsenic?

THE DIAGNOSIS AND TREATMENT OF ABNORMAL UTERINE BLEEDING*

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THIS discussion is limited to the significance of various types of pathological uterine hemorrhage and excludes that associated with late pregnancy and labor.

To appreciate the abnormal we must accept a working hypothesis of normal menstruation and pregnancy. Much research still leaves many points at issue. For practical purposes, however, we are justified in certain dogmatic assumptions based on the work of Frank and his collaborators.

1. We assume the formation of a single, specific "sex hormone" secreted in the ovary; certainly in the Graafian follicle and corpus luteum; and probably also in atretic follicles, stroma, and the so-called interstitial cells. After implantation of the fertilized ovum, this hormone is stored or formed in the decidua and placenta.

2. The relatively small amount of hormone furnished by the immature ovary, in conjunction with internal secretions of the pituitary, thyroid, adrenal, and other glands, is sufficient to determine prepubertal development of the special and secondary sexual characteristics.

3. Periodic maturation of the ovum, including development of the Graafian follicle, its rupture, and formation of the corpus luteum, all of which begin at puberty, causes recurring waves of increase in the amount of sex hormone in the body, followed by sharp decline. These cyclical variations induce equally constant changes in the uterus and endometrium, sufficient to provide a suitable field for implantation of a fertilized ovum, should it be present. In that case the corpus luteum persists for a time. From this source and from the decidua and placenta, an adequate amount of hormone is provided to maintain a status quo in ovaries and uterus to nourish and to protect the growing product of conception. It prevents uterine bleeding and probably inhibits ovulation.

In the absence of implantation, there is a sharp decline in the amount of sex hormone, associated with atrophy of the corpus luteum and bleeding from the congested endometrium. Therefore, physiological uterine bleeding, menstruation, occurs only from an endometrium altered by an increase of sex hormone associated with ovulation, and is due to a sudden decrease of this secretion in the absence of successful implantation of a fertilized ovum. A number of factors modify the duration and amount of hemorrhage. Variations in periodicity are always pathological.

Menorrhagia, excessive or prolonged menstrual bleeding, is a relatively abnormal reaction to normal stimuli. It is pathological when the blood lost is not restored during the intermenstrual period. Anemia is frequently present and indicates the severity of the condition. Following puberty some variation is common. Usually it is temporary and self limited, being of slight significance. In other instances constitutional treatment is followed by readjustment. An occasional case, however, will present serious effects and prove most baffling.

Endocrin imbalance, blood dyscrasia, and, rarely, local lesions are to be considered in the etiology. Endocrin factors include ovary, thyroid, pituitary, adrenal, possibly other glands. The disturbance is often temporary. Gland therapy is still empirical or worse, assurance of detail men to the contrary notwithstanding. Blood dyscrasia, hemophilia, and thrombocytopenic purpura are surprisingly frequent in young women. Low blood platelet counts, under 60,000, are suggestive. Chlorosis, with its characteristic blood picture, is not uncommon. These conditions are the cause of many obstinate cases of menorrhagia. Therapy with calcium, iron, or blood transfusion is most effective. Tuberculosis and lues are occasional causes of this type of bleeding.

X-ray treatment of the splenic region through its action on blood formation has some value. Effort to modify ovarian secretion by this agent, unless used with great skill together with a large measure of good luck, is likely to do more harm than good. At this period of life some type of constitutional treatment is the method of first choice. Local pathology is less common and less important than later in menstrual life. Pelvic

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examination under anesthesia is usually negative. Uterine curettage is rarely of therapeutic value. Resection of ovarian cysts or a cystic ovary may occasionally give more than temporary improvement. Hysterectomy or castration in the young is a frank confession of failure. However, one may be forced to consider it in uncontrollable cases.

The importance and frequency of local causes increase with years, with infections, and with childbearing. Tumors, local inflammation, subinvolution and prolapse—all predispose to menorrhagia. Thorough examination will reveal the cause. Properly selected surgical treatment will control the bleeding, and retain ovarian secretion. Hence it is truly conservative. In some cases of prolapse and subinvolution subtotal hysterectomy offers the most effective solution. Radiotherapy in a carefully restricted group of cases may be considered. Contraindications are: Inflammation or sepsis, ovarian tumor, large uterine tumor, submucous fibroid or polyp, cachexia or severe anemia. Malignancy must be excluded. Pregnancy must be interdicted for at least a year following treatment.

Metrorrhagia or irreguar uterine bleeding of whatever degree is an objective sign of serious import. It is independent of ovulation. It is always abnormal. The amount may vary from blood-tinged discharge to profuse and dangerous flooding. It may follow local trauma, examination, douche, or intercourse. It may be intermittent, or nearly continuous. In any case one must exhaust every means to find the cause and carry out proper treatment without delay.

We shall consider two groups.

1. Bleeding due to a pathological condition in early pregnancy.
2. Bleeding due to other local lesions.

Any bleeding during pregnancy is pathological. It may occur before there is reason to consider pregnancy. If accompanied by unilateral pain, one must suspect an ectopic implantation. So-called menstruation during gestation is due to atypical hypertrophy of the decidua which bleeds easily in response to variations of the sex hormone. This bleeding is periodic in type, slight in amount, unaccompanied by pain. It is quite different from that associated with placental separation or threatened abortion. Thyroid extract is of definite value.

Impending or actual abortion with disturbance in placental attachment is the most frequent cause of uterine bleeding during early pregnancy. In such cases one must consider several questions:

1. Is the abortion only threatened or is it inevitable? If the blood lost exceeds that of normal menstruation, or if there are severe uterine contractions, the fetus is probably dead and will be expelled. Rest and narcotics may prevent threatened cases, but do not delay an inevitable abortion.

2. Is the interruption of pregnancy spontaneous, or has it been artificially induced? In the latter case we recognize an increased liability to infection and to retention of secundines.

3. Is the abortion complete? Examination of the material expelled may give the answer. Bleeding continues till the uterus is empty. Unless this has been accomplished spontaneously within a few days or a week, mechanical assistance is necessary. Oxytoxics are uncertain but may be tried. Vaginal manipulation or packing is but little safer than proper exploration under anesthesia. It is also of slight value. Hemorrhage is the only indication for interference. It is rarely sufficiently alarming to justify emergency operation. One must allow time to exclude infection. History of previous vaginal manipulation requires extra caution. Repeated intrauterine operations should be unnecessary; they are dangerous as well as useless. An adequate procedure will control bleeding in all simple cases, or suggest the presence of other conditions. Fever means infection and contraindicates any local manipulation. Study of the curettings may disclose hydatid mole or malignancy. Negative uterine findings together with careful bimanual examination will sometimes disclose an ectopic pregnancy which is in process of abortion. The clinical picture is often confusing. Uterine bleeding begins with disturbance in placental attachment and continues till ovulation is re-established.

Aside from pregnancy, metrorrhagia is always due to local lesion with destruction of the endometrium. Its causes include:

1. Preclimacteric hypertrophy of the endometrium, in rare instances. Diagnostic curettage fails to disclose cancer. This operation, together with suitable pelvic repairs, is followed by cure. In case of recurrence the presence of cancer must be assumed and radical treatment instituted.

2. Submucous or pedunculated fibroid tumors are associated with hyperemia and destruction of the endometrium resulting in metrorrhagia. Di-

agnostic curettage and biopsy is necessary to exclude cancer which may accompany gross tumors. In its absence excision of the growth or hysterectomy is the proper procedure. Radium is not advisable.

3. Cancer involving the mucosa promptly causes intermittent bleeding. This is the earliest and most constant sign of the disease. This type of abnormal bleeding, including postclimacteric hemorrhage of any sort is an absolute indication for immediate diagnosis and treatment. Biopsy and study of curettings permit the diagnosis of cancer. If properly done it is without danger, provided therapy is instituted at once. Study of cell structure will influence prognosis and type of treatment. For cancer of the body or fundus, total hysterectomy offers a good prognosis, and is the procedure of choice. For cancer of the cervix or vaginal portion, radium, properly applied, has largely supplanted surgery, even in early operable cases. Systematic follow-up with efficient doses of deep *x*-ray is advised.

To summarize: Variation in amount or periodicity of uterine bleeding is a frequent symptom and sign. Abnormal duration or amount indicates a study of constitutional factors, after which local causes should be sought. There is comparatively slight danger from reasonable delay for investigation. Variation in the usual periodicity, however, points to local pathology. Diagnosis is urgent and is always possible. This must precede treatment. Curettings must be studied grossly and microscopically. There is no excuse for repeating a therapeutic curettage. We must emphasize to ourselves and to our patients the fact that metrorrhagia or post-climacteric bleeding is always pathological. It is only by full appreciation of this fact that we can reduce the mortality of cancer of the uterus.

Again, abnormal uterine bleeding is an important objective evidence of disease, unequivocal in character, and simple in interpretation. Let us give it the attention which it deserves.

HYPERTHYROIDISM IN CHILDREN*

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UNTIL a few years ago hyperthyroidism in children was thought to be extremely rare. Although the condition is being met with more frequently, we can still follow Helmholtz' dictum that it is extremely rare in early childhood, and becomes more common with the approach of puberty. It seems to be, however, more or less limited to the female, for I am able to find only one case in the literature of hyperthyroidism in a male child under ten years of age.

In agreement with previous articles on this subject we have chosen fourteen as the upper age limit. Although this series of eight cases is rather small, still the lessons learned in handling them have been very interesting and instructive.

REVIEW OF LITERATURE

Lewis in 1914 reported five cases of exophthalmic goiter in children among 1,512 goiter patients operated on at the Mayo Clinic between 1905 and 1913. Sattler has reported the largest series, having studied 184 cases in children among a total of 3,477. Dinsmore in 1926 reported forty-eight cases of hyperthyroidism in children observed at the Cleveland Clinic, eight being in boys and forty in girls.

Numerous single cases have been reported by various men, while White has reported a case of exophthalmic goiter in a newborn infant where the mother was suffering from the disease, and Klaus in 1914 reported a case in a nine month old infant.

ETIOLOGY

The cause of this disease is more or less uncertain. However, by reviewing the case records of the various individuals coming under my observation, I have come to some definite conclusions as to the factors which to me have been practically specific in causing a hyperactivity of this gland.

Infections.—Three of the patients in our series definitely attributed the onset of the present illness to some acute infectious disease, two of them having had influenza and one scarlet fever within two years of the time they came under observation. Many other infectious diseases such as tonsillitis, sinusitis, and pneumonia, have been reported as factors in the production of hyperthyroidism.

Shock, Injury or Emotional Disturbance.—A great deal has been written and there have been many discussions regarding the influence of bodily injury or mental shock on the activity of the thyroid gland. This discussion has been confined mostly to adult cases, and from a medico-legal point of view a great many theories have been advanced; however, very little has been said regarding this factor as a causative agent in children.

Hill reports a case, that of a seven year old girl, who, on account of some harrowing experience encountered during the war, developed a typical exophthalmic goiter.

In our series of cases, there were two which we can definitely place in this category. One patient had been slightly injured and badly frightened while on a sleigh ride. The other, a girl, had been perfectly well until the afternoon her grandmother was buried, when she suddenly became rather hysterical and began vomiting, which was followed by the typical hyperthyroid syndrome.

Use of Prophylactic Medication.—This is another phase which brings forth a great deal of discussion. So much has been written in the newspapers and lay magazines regarding the prevention of goiter that a large number of so-called preventatives have been placed on the market and given to children with absolutely no thought of putting the child under the care of a physician while the medication is being used. It is rather left to the grocer, druggist, nursemaid or mother to determine the dosage. While I believe the prophylactic use of iodine or one of its derivatives is a perfectly correct procedure when given under medical supervision, nevertheless we have seen cases of hyperactivity of the thyroid gland develop in such instances. Marine and Kimball in their series of 4,415 children who received iodine as a prophylactic had from 0.5 to 1 per

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cent of their cases develop symptoms of hyperthyroidism which promptly disappeared when the medication was stopped.

DeQuervain had a similar experience, but thinks that the risk is nil if the prophylactic dose does not exceed three milligrams.

Bram in a series of 3,343 cases saw 3 per cent develop hyperthyroidism while an additional 1 per cent had trouble from the use of thyroid extract.

By far the most dangerous prophylactic used today in my opinion is iodized salt. Three of the patients in our series gave a definite history of having used this type of salt from two to three years, and without doubt this changed an inactive goiter to a very active type.

Adolescence.—Because of the rather sudden rise in the number of cases of exophthalmic goiter about the time of puberty, the theory has been advanced that the change in the life cycle may be a factor in causing an increased thyroid activity. The great majority of cases of hyperthyroidism, as we know, occur during the period of sex life, and it is a very reasonable theory that the ovarian activity has an inter-relationship with the activity of the thyroid, and vice versa.

PATHOLOGY

The glands in this series of cases (and all were partially or totally removed save one in which a unilateral superior lobe ligation was done) showed the gland to be bilaterally enlarged, rather firm in consistency, with some areas throughout the gland which were soft and friable, evidently areas of degeneration. Such an area may often on palpation be confused with a small adenoma, which would follow Kitchen's observation on examining large groups of children having enlarged thyroids. None of these patients had true adenomas, and I am unable to find any case in the literature of hyperthyroidism with adenomas in children. In fact Plummer states that it is an exceedingly rare observation in persons under the age of twenty-five years.

The microscopic picture was somewhat varied in that certain portions of the glands showed a true hyperthyroid state with cells of the columnar type, some of which were undergoing mitosis, with the acini small, and filled with a rather small amount of thin material. On the other hand portions of the gland gave the appearance of being inactive with the cells flattened, the acini distended with colloid and a marked decrease in the vascularity. Whether or not this is the picture that persists during the disease or whether this colloid portion of the gland is due to the administration of iodine in the pre-operative treatment, is a matter of speculation, but I believe it is highly plausible that iodine medication bears a part.

SYMPTOMS

In the past it has been the consensus of opinion that hyperthyroidism in children has a rather prolonged onset or course with the various symptoms coming on slowly, the duration of the symptoms varying from one to twelve years; in other words that acute hyperthyroidism in children is a rare observation.

This is undoubtedly true, nevertheless one of the patients in this series dated the onset of her illness to twenty days previous to her admission to the hospital, and at that time she was in a true crisis.

Carroll reports the case of a girl thirteen years of age, who was in an accident, at which time she experienced a severe fright. She developed a true exophthalmos with symptoms of exophthalmic goiter within twenty-four hours after the accident. The symptoms increased in severity and she made an uneventful recovery following a thyroidectomy, preceded, as was the usual case some years ago, by bipolar ligation. From these two cases we see that hyperthyroidism may have an acute onset.

The illness usually begins with marked nervousness and irritability, and following this the parents usually observe an hypertrophy of the gland.

Tachycardia was present in all the cases in this series, ranging from 120 to the most severe case, in which the pulse was persistently 175. The manifestation of tremor was not constant. Only one girl showed a true tremor. Weight loss was almost a constant observation, ranging from five to fifteen pounds.

Helmholtz believes that this type of case should be called true exophthalmic goiter. Nevertheless not all of these cases develop the typical ocular changes seen in this type of goiter. In fact this is a rare observation in some series reported, although Dinsmore found a large percentage of his cases with true exophthalmos.

In our series four patients showed marked

exophthalmos, one a slight degree, and three had no eye changes. All of these children showed a normal mentality; in fact the majority showed a tendency to be beyond their years mentally.

Just how accurate the basal metabolism readings are in these cases is questionable. Whether or not a way will be worked out to determine this reading more accurately remains to be seen. The rates in our series varied from plus 38 to plus 178, but I believe that these are all higher than they should be. The fact is the child with a rate of plus 35 or plus 40 may be considerably more hyperthyroid than a plus 75 or 80 when we compare the rest of the clinical picture.

TREATMENT

Treatment of these cases was that usually carried out in exophthalmic goiter, but there are two or three points that I wish to emphasize. First of all the preoperative preparation. As in the adult, Lugol's solution was our greatest single weapon of attack. However, there is a great difference in the time element necessary in the preparation of these cases. We know that the ordinary adult with exophthalmic goiter can be prepared for a thyroidectomy in from twelve to twenty days under Lugol's solution, but this is not the case in children. A great deal longer is required or the patient will not withstand any surgical procedure, no matter how slight. Two cases have been brought to my attention, the one having been prepared with Lugol's solution for twenty-one days and the other for sixteen days. In the first case a one sided lobectomy was done and in the second a ligation, but immediately after operation each patient developed a true crisis and died within twenty-four hours. From these cases I believe that instead of days for preoperative treatment, we must take weeks and often as high as three months.

As far as the operative technic is concerned, I believe it is difficult to be too radical in removal of the gland because of the tendency of the small remaining portion to rapidly hypertrophy.

Although outside the scope of this paper, I have seen two girls, fifteen and sixteen years of age respectively, who have had to undergo a second operation within a year's time for the removal of a rapid recurrence of an hypertrophic gland.

As in adults, I believe that Lugol's solution should be given post-operatively for a varying length of time, and that these patients, if possible, should be kept under observation for at least one year after operation.

SUMMARY

Because of the inconstant finding of ocular changes, I believe that this type of disease should be called hyperthyroidism rather than exophthalmic goiter, and that it is a much more common disease in young girls than previously thought. In the large majority of cases we find that some infectious disease is an important causative factor, and that shock, injury, fright, and promiscuous use of iodine as a prophylactic are very important etiological agencies. Symptoms are usually comparable to the adult type but the basal metabolism reading is not an accurate gauge of their true condition. A much longer pre-operative period is required than in the preparation of adults, and radical removal of the gland is the only possible cure.

THE INDUSTRIAL EYE*

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THE rapidly increasing use of automobiles and aeroplanes together with the widespread improvements in roads, bringing high powered machinery into the outermost parts of the country, has brought the question of the Industrial Eye to every physician, no matter where located nor what line of work he may be pursuing. It is, therefore, our belief that it will be time well spent to go over, somewhat carefully, the present understanding of this most interesting subject.

In the first place there is one consideration in every eye case which is often forgotten or overlooked. That is, the vision of the injured eye at the time the patient presents himself. Often this is overlooked because of an apparent mild injury or an outlying foreign body, and later the patient returns with a report of loss of vision, when the record of a visual test at the time of accident would have saved great expense to some one for compensation or damages. It is also needless to say that money would be saved to employers, should all applicants for work be given a preliminary visual test and careful record made therefrom.

The next step is the history, if it can be obtained, of the accident or of the development of the complaint. This should be taken very carefully, as we have had foreign body cases to be charged to the Company while the man was working on his own car during working hours.

Having obtained this information, the eye should be given what we are pleased to call the external examination. Every trained eye man has spent many hours going over the external examination of eyes very methodically until at a glance he can estimate where the pathology begins and is in position to make a complete record up to this point. To illustrate: An external examination proceeds as follows:

1. The general appearance of the patient's face and the skin about the eyelids is examined for evidences of injury.

2. Lid margins are examined, openings of the various glands, then condition of the puncta followed by compression of the lacrimal sac showing presence or absence of chronic infection there.

3. The conjunctival sac is thoroughly inspected as to:

   a. That portion covering the under sides of the eyelids.
   b. That covering the globe.
   c. That in the fornix, above and below.

The cornea is next thoroughly inspected as to smoothness of its surface, clearness and evidence of previous injury as well as evidence of present injury.

The anterior chamber is inspected through the cornea and its depth noted as well as the character of the aqueous.

The iris is next noted as to its integrity as well as activity.

Finally the lens is inspected, as well as the inner contents of the eye and the fundus.

Following this the finger tension is taken before any atropine is instilled.

If there is a lack of visible injury of the eye the muscle excursions are tested.

After making this examination, the evidences of injury are now weighed and an estimate of the immediate seriousness is completed before removing a foreign body or repairing an injury. Based upon these estimations a prognosis is then made as to the possible duration of disability and as to the probable outcome.

Having accomplished this, the eye is then anesthetised, foreign body, if one is present, is removed, or removal is attempted. The eye is then given a second examination following this to recheck the amount of damage done. Under all ordinary conditions this is followed by placing the eye at rest and as a rule covering it for a period of from twenty-four to seventy-two hours, as judgment indicates.

With the patient ready to be discharged we then, again, estimate the present amount of vision, comparing it with the vision taken: (1)

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at the time of injury; (2) at the time of employment, if that has been done.

As you are well aware we have several tables estimating the loss of vision, none of which are ideal but all of which are reasonably accurate. It is our opinion that the table (see below) prepared by the Minnesota Academy of Ophthalmology and adopted by the Oliver Iron Mining Company and State Industrial Commission is of the best at the present time, although Dr. F. Park Lewis of Buffalo and other men with vast experience in this kind of work are now attemtping to provide a new table.

TABLE OF VISUAL LOSS ADOPTED IN MINNESOTA
(Minnesota Academy of Ophthalmology)

READING	PERCENTAGE OF LOSS
20/20	Normal
20/25	5
20/30	10
20/40	20
20/50	25
20/60	33
20/70	40
20/100	75
20/120	85
20/80	50
20/200	100

Before proceeding to cite cases may I say that it is our belief that all cases of industrial eyes not showing improvement from any condition in a period of twenty-four hours following first examination should be referred to a trained oculist.

What then should be the picture in first aid in an Industrial Eye?

1. A visual test if possible.

2. An external examination looking for foreign particles in the skin of the lids, conjunctival sac, cornea, etc.

3. Cleansing of conjunctival sac of foreign material, using no rough or irritating methods, nor strong antiseptics. We prefer and recommend the use of normal saline, warm, or occasionally 1-5000 cyanid of mercury used after the instillation of a local anesthetic.

4. Removal of superficially attached foreign bodies from the cornea or conjunctiva. Any foreign body involving the stroma should be removed with great care, as injury to the stroma always results in scarring of the cornea with some loss of vision.

5. Instillation of one drop of one per cent atropine in the eye except where the tension of the eye is up or where past history is suspicious of a glaucomatous increase in tension.

6. Referring of any severe or even suspicious case to a trained eye man at the earliest rather than a late date following injury. Insurance companies never dislike to pay early bills but they frequently do question late bills.

205 Power Bldg.

PSYCHOANALYSIS IN REACTIVE EPILEPSY*

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SINCE you are all familiar with epilepsy, you know that if we disregard the classifications which are based on the description of the epileptic attacks, all cases fall into two broad groupings: those cases in which epilepsy is symptomatic of cranial disease, and those in which no adequate brain pathology can be found and held accountable for its production. Of the latter type, which is the idiopathic or genuine epilepsy, there are certain cases in which there is present a marked disturbance of the emotions, sensibilities or feelings. There are many ways in which the emotions of this particular group of epileptics differ from those in so-called normal individuals, but the most common ones are: (1) the existence of an irritable tenseness in the interval between the attacks, which, though it may not be constantly displayed, is always very close to the surface; and (2) the likelihood that any violent emotion in the nature of anger, fright, fear or hate, may precipitate the convulsive or petit mal seizure.

The convulsive reaction of these patients to such emotional stress led Bonhoeffer to coin the term "reactive epilepsy," or to use a broader term which suggests greater involvement of the feelings or affects, Bratz uses affective epilepsy. No matter what term is used, it is merely one of description and of little consequence. The important thing is that in certain epileptics there is an abnormal emotional condition which must have its origin in the mind.

The forces which contribute to the faulty emotional state are partly within and partly beyond the scope of psychoanalysis. Within its compass are the complexes, repressions and ideals of the individual and beyond it are the personality or character of the individual and the ever present difficulty in epileptics of adjusting themselves to their family, social and economic obligations. Yet when you consider it, character and social adjustment are essentially mental reactions and are certainly influenced by our experiences. As an instance, let me say that egoism and hypersensitiveness are the dominant traits of character in the epileptic, but hypersensitiveness and egoism are interdependent because the egocentric person's interests are so narrow that they are constantly considering every contact and event in relation to themselves, and it follows that those contacts which are not advantageous to them are looked upon as slights.

This is how one patient explained the difficulty: "Even as a child I was always self-centered and self-conscious. Everything that happened seemed to travel in a circle which began and ended with my benefit in view. I struggled against the narrowness of this outlook because as I grew older I realized that my feelings were being hurt by everything that my family or friends did or said which did not definitely take my likes or dislikes into consideration."

While this view explains their sensitiveness, the egocentricity of their nature has deeper and more fundamental aspects. Some of the factors which contribute to it are, the prominence which the child assumes in the affection of its parents between the ages of one and three years; the example of egoism as a characteristic of one or both parents; certain methods of correction which make the child irritable and nervous; sexual desires and repressions in infancy, and too much praise for minor successes in early childhood. All of these factors, and many others, contribute to the child's egocentricity, and tend to bring about in the child hypersensitiveness and imaginativeness, because of the emotional involvement, and also to make it more or less tense and fearful, a typical foundation for a nervous disorder either early or later in life.

Let me report a case which developed epilepsy in her thirty-first year:

From her earliest recollection she was apprehensive, imaginative and nervous. Very early in childhood she had difficulty in sleeping; lying awake she was possessed with the fear that she would be stolen by gypsies and lost. From this fear she developed a horror of the color red, which made up the head dress and jacket of the gypsy of her imagination. Her emotions were always very near the surface, but in childhood a morbidity manifested itself and her memory now leads her to think that she spent most of her youth longing for death. She wet the bed until she was seven years old but had no other suggestion of

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epilepsy, excepting that she was shy, self-centered, emotional, and displayed a typical nervous tendency. At the age of twenty-two years her father met with financial reverses, and she had to work to help support herself and contribute to the family, and here, for the first time, it was necessary for her to make a number of adjustments in her life. She had no business education but more than the average intelligence and she entered a large office as a clerk. Within a year she was assistant to the head of the department, and as her salary increased she began to contribute to the education of a talented older sister. As she bettered her position she increased her responsibilities, so that at the age of thirty-one she was supporting herself, her mother and father, a younger brother who was in college and contributing to the support of two sisters who were studying music in New York. This burden does not suggest a selfishness of character, and yet the selfishness was present in her in a very great degree. Her personality was lively, vivacious and clever, but underneath this veneer she was cruel and ruthless. Her family held absolutely no place in her affections. She was entirely devoid of love for any of them, and still she had been supporting them for years because her mother, with uncanny accuracy, had played upon her egoism, and this by the rankest kind of flattery. The girl was wonderful, the mainstay of the family, their only support; she was worshipped, adored and palavered over incessantly. While she was entirely conscious of the fact that this was a small return for her sacrifice, still she was unable to withdraw from the situation.

This brief review of her family situation shows at least some of the difficulties which beset her, but the analysis brought to light another phase of her life.

Let me relate one of her dreams: She was walking on a hill somewhere near Seven Corners in St. Paul; on this hill the buildings flank the sidewalk and in it are many windows almost at street level. As she went down the hill she found the street blocked with a great crowd of horses which frightened her half to death. As she ran back up the hill there were many horses in each window of the building. She ran to a livery stable at the top of the hill and a man came to the door and told her she might go through the stable and out the back window, which she did. She climbed down a ladder, like a fire escape, to the street. When she finally reached the street and looked up the hill, all of the horses ran down the hill after her.

In this dream the motion, horses, horses' heads through windows and the escape and pursuit have a sexual significance. After the psychoanalysis was under way for about a month, she had related seven other dreams in which horses jumped on her, ran after her or frightened her in some other way.

This repetition in dreams is thought to have its origin in some traumatism and the following was the incident which gave rise to her horse dreams:

At the age of 23 years a man by whom she was employed part time finding that they were alone locked the office door and attempted to have his way with her. She struggled and then fainted. As she recovered consciousness, certain impressions of him made her think of him as a horse. This incident was so painful in her consciousness that she repressed it to the point where she had forgotten all about it, and, in addition, she had repressed all sexual desires by creating in her mind a hatred and contempt for all men.

This sketch then serves to add two more factors to her family and economic trials: those of traumatism and sexual repression. It is impossible to detail the effect that the necessity for adapting herself to these various situations had upon this girl, but as she bore them all she became increasingly irritable and tense. This displayed itself in a certain impatience with her work; with people who came in contact with her; in a feeling that she must get her work "done up" and by the rather stubborn tenacity with which she drove herself to accomplish her tasks. However, she carried on for about eight years, before she developed her convulsions.

To conclude this case: after explaining her situation to her and removing some of the mental difficulty and economic stress, this girl was free from convulsions for eighteen months, when she was lost from my observation.

As has already been said, epileptics are egoconscious and in the mind of each one of them there is a tangled mass of psychopathic material which promotes the difficulty they experience in adapting themselves to social and economic circumstances. According to L. Pierce Clark, a widely quoted author on the subject, the lack of social virtues, kindness, charity, love in the social sense, generosity and consideration for the feelings and rights of others, makes it difficult or impossible for them to make a social adjustment. The magnitude of their hypersensitiveness and egocentricity, combined with the degree in which the social qualities are lacking, he believes determines when, that is what time in life, they will develop their epilepsy. Those people who are very egocentric and hypersensitive and who are almost completely lacking in adaptability will develop it early in life, even if they are subject to little or no conflict with reality, while those people who have less pronounced character defects will only develop the disease when the stress of adapting themselves becomes relatively great.

While this theory of Clark's is attractive, no attempt is made to understand why epileptics should develop such a character, other than to throw the whole problem back to the unalterable factor, heredity, that is familial left-handedness, alcoholism or syphilis in the parents, hereditary burdening or such a vaguely descriptive term as the neuropathic diathesis.

In this communication no pretext is made of having solved the entire problem of epilepsy, but

the method of approach is somewhat different and it at least allows us to understand some of their reactions and ideals. Let me give an example:

An intelligent young man has had a reactive epilepsy for a number of years; it began at the age of nine. He has a vast psychopathic background, which contributes an emotional difficulty to his life. The stress in his life is briefly as follows: Since he graduated from school he has been employed by and supervised by his father, who is not only a hard taskmaster but has insisted on the young man assuming the duties of salesman, laborer, book-keeper, gardener, housekeeper and various other jobs, many of which were in competition with other healthy men. This competition, supervision, and the closeness of the family contact necessitated by his condition, has contributed to his irritability. However, to keep a certain amount of peace in the family, to hide his condition as much as possible from outsiders and to please people in business, he has assumed a certain unctuous, suave manner. The unctuousness is, of course, entirely assumed and words of endearment are plentifully scattered through his conversation, but his whole manner is so unconvincing that his efforts are clearly transparent and his effect shallow. This same man is exceedingly loquacious, bringing dates, names and circumstances into the conversation with great freedom. The purpose in this display is to convince himself, as well as his hearer, that there is nothing wrong with his mind.

Attention is called to this mechanism so frequently employed by epileptics because the literature on this subject describes the shallow feeling, the unctuous emotionalism, the superficial religiosity of the epileptic. The impression which their display of emotion makes on the observer is certainly superficial but it must be understood that their manner is purposely assumed to help them to make a social impression, and to cover up their real feelings. The very fact that they are so sensitive to the impressions which they create, and so fearful of criticism, makes them self-conscious and less able to carry off their role. If they begin to deteriorate mentally, the role they have assumed may deteriorate likewise until their utterances are disjointed phrases, religious, endearing or of superiority.

In the last case cited, reference was made to the manner in which he reassured himself of the integrity of his mental functions, and this suggests the idea that his hypersensitiveness also embraced his own body and mind as well as the impression he created in other people. In other words, was he critical of himself, and, if so, would the irritation which he felt with himself have any influence in producing the attacks? He was placed on relatively large doses of bromides so that his mental processes were decidedly slower. After the second day's administration he became very irritable. That night he had eneureses and continued to have it as long as the bromides were given. With the stopping of the bromides and the resumption of his normal mental activity, the eneuresis stopped. Whether the eneuresis was accompanied by a convulsion, or was a convulsion, cannot be told, but it may be assumed that it was one, or its equivalent.

This instance would seem to indicate that irritation arising from self-criticism was sufficient to bring on certain seizures, and further that he must have a certain ideal in regard to the manner in which his ego functioned. So, viewed from almost any angle, the emotions of the individual and their expression for the most part by irritability would appear to arise from a common source, the ego, and by ego is meant the individual's appreciation of self. If the whole fabric of epilepsy reverts back in every instance to egocentricity, then it would appear from the psychoanalyst's view, as has been asserted by Freud, that nothing can be accomplished by that method. Freud believes that the ego is a fixed point and that psychoanalysis can only be concerned with subconscious or unconscious matters which circulate about this fixed point.

This conception of Freud's is undoubtedly an artificial distinction made for teaching purposes, for a moment's consideration will convince you that egocentricity is a variable, which changes with a number of things. The invalid, for example, is more egocentric than the well person, but with the return of health the egocentricity largely disappears. This being true, the invalidism of the epileptic may determine to some extent his egocentricity, and so, even if Freud's "fixed ego concept" is true, egocentricity, or the degree of self-appreciation, is a variable quantity, and the causes for it lie within the scope of psychoanalysis. Psychoanalysis will remove some of the psychopathic mechanisms present and increase our understanding of their personality and their moods.

THE PHYSIOLOGY OF SPINAL ANESTHESIA*

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WITH the recent popularization of spinal anesthesia many attempts have been made to explain the various phenomena which take place. It was with the idea of correlating the explanations of these phenomena that this paper was written.

In spinal anesthesia or subarachnoid block, a block is produced which prevents impulses being conducted through the anterior roots to the spinal nerves, or in the reverse direction from the spinal nerves through the posterior roots to the spinal cord. The anesthetizing drug, novocain for example, sets up a chemical reaction with the nerve tissue producing a physiological block. This block occurs at the anterior and posterior roots of the spinal nerves within the dural sac, because here they are more sensitive to and more easily permeated by the drug.

Not all of the fibres in the nerve roots are similarly affected. Some fibres are more resistant than others. Evans[2] states that the fibres are blocked in the following order:

1. The fibres which carry pain and temperature sensations from the skin and viscera.
2. Those fibres which arise from the skin and convey the sensation of touch.
3. The fibres which carry impulses of pressure or movement from muscles.
4. The fibres which carry impulses to the skeletal muscles.
5. The sympathetic nerve fibres which receive stimuli from the viscera and send out, among others, impulses which regulate the vasomotor system.

Thus, one can see that those fibres having to do with the sensation of pain are most affected, while those having to do with vasomotor control are least and most fleetingly affected. Therefore, in spinal anesthesia, there is first loss of the sense of pain, then of tactile sense, then motor sense, then of muscle power, and finally of vasomotor control. As the depressant or blocking action of the drug wears off, conduction through the nerve fibres gradually returns to normal and the lost functions return in the reverse order.

The anterior nerve root carries the motor fibres, while the posterior nerve root carries the sensory fibres. The posterior or sensory root is more readily affected by the drug, thereby producing loss of pain, temperature and muscle sense, while the anterior root is affected to a lesser degree. Besides this, there are several factors which favor a more concentrated solution upon the posterior roots. The first of these is the point of injection. The anesthetizing drug is injected posteriorly and therefore must necessarily come in contact with the posterior roots first, and will be more dilute when it reaches the anterior roots. Secondly, the interarachnoid trabeculæ, between the arachnoid and pia mater, hinder to some degree the diffusion of the drug anteriorly. Lastly, the position of the patient, which is on the back, and the specific gravity of the injected solution, tend to keep the more concentrated solution about the posterior roots.

There are certain phenomena which accompany spinal anesthesia that are of considerable interest. One of the more important of these is the fall in blood pressure. There are three factors concerned here. The first of these is the loss of vasomotor control. Normally there is a balance set up between the vasoconstrictors on the one hand and the vasodilators on the other. This balance is regulated by the vasomotor center. Now, if the vasoconstrictors, which tend to keep up the blood pressure, are paralyzed, then the vasodilators are overactive, the blood vessels dilate, and the blood pressure goes down.

Experiments on animals have shown that, as the spinal cord is cut, various drops in blood pressure occur, depending upon the level at which the cord is severed. For instance, cutting the cord at the level of the first thoracic

*From the Duluth Clinic, Duluth, Minn. Read before the Minnesota Surgical Society, Duluth, Minn., Sept. 14, 1929.

segment produces a maximum lowering of blood pressure. A mid-thoracic section would produce a less marked drop, while a section through the lumbar region would result in very little or no drop. The same mechanism is involved when spinal anesthesia is administered, and the degree of drop is determined to a large degree by the height of the anesthesia. In practice, this drop in blood pressure is overcome in some degree by the administration of ephedrin[6] or adrenalin, which act peripherally to stimulate the vasoconstrictors, and thereby maintain or elevate the blood pressure. Many cases* maintain the blood pressure at the normal level; others will show either a fall or a rise of ten to twenty millimeters, and only occasionally is the drop sufficient to cause any alarming symptoms.

The second factor involved in the blood pressure drop is the paralysis of the thoracic and abdominal muscles. By this paralysis the pumping or aspirating action of the thorax is lost or diminished and a smaller volume of blood gets to the heart. In other words, the minute volume is decreased.

The third factor is the slowing of the heart rate. Here again there are two nerve supplies which are antagonistic in their action—the vagus or inhibitory nerve, and the sympathetic or accelerator nerves. The vagus arises from the medulla and soon becomes extradural. The sympathetic nerves arise from the first to the fourth thoracic segments, and pass out as part of the anterior nerve roots. Thus, if the anesthetizing drug reaches these nerve roots there will be a block, and the amount of block will determine the amount of slowing of the heart rate. The vagus will become overactive and the heart rate will be decreased.

Another interesting phenomenon which is met with is that which, for want of a better term, is called "increased peristalsis" or "contracted gut." The same mechanism which explains the drop in blood pressure and the slowing of the heart rate will explain to some degree this last phenomenon of contraction of the intestine. In this case the sympathetic and the parasympathetic systems are again antagonistic. The sympathetic fibres arise from the lower thoracic and upper lumbar segments, while the parasympathetic impulses are carried through the vagi and the pelvic nerves. The vagus supplies the small intestine, the ascending and transverse colon, while the pelvic nerve supplies the remainder, *i.e.*, the descending colon, sigmoid, rectum and anus. The sympathetic fibres exercise an inhibitory action on the intestines and the parasympathetics a contraction. The fibres which make up the pelvic nerve are always paralyzed in a spinal anesthesia, as it arises from the sacral roots, so one can see why we have a contracted gut and a relaxed anal sphincter. There is a partial or complete blockage of the sympathetics, allowing the vagus and the intrinsic nerve supply to become overactive, resulting in increased peristalsis. Whether or not the antagonism between the sympathetic and parasympathetic nerves is as definite as it would seem here is doubtful. However, Alvarez[1] states that the first effect of stimulation of the vagus is generally a slight lowering of the tone of the stomach and bowel, and this is followed in a few seconds by an increase in tone and activity which may last for from a few seconds to a minute. He goes on to state that Spodolini and others have shown that one can get purely inhibitory effects by stimulating the vagus, just as one can get purely augmentatory effects by stimulating the splanchnics. Ordinarily the main effect of stimulating the splanchnics is an inhibitory one. Certainly, at least, the antagonism does not seem to be as definite as in the vasomotor system.

Lapicque[4] introduces a vital and novel theory to explain not only inhibition and anesthesia but fundamental selectivity in nerve reactions. According to his theory one neurone system can stimulate another only if the "temporal characteristics" of both are equal or nearly so. This "temporal characteristic" harks back to a hookup of nerve connections based not so much on direct continuity as in a set of electric bells or a telephone system, but rather as a "tuning in" arrangement such as obtains in radio. On such a basis any local or general anesthetic need not so much mechanically deaden pathways of sensation as to close them by changing their "vibration," "wave length," or "tuning in" properties.

If one eliminates the reflex nausea and vomiting brought on by manipulation of viscera

*Personal observations based on 600 inductions of spinal anesthesia for operations below the diaphragm.

in the upper abdomen, especially the stomach and duodenum, there remain two other factors which produce nausea and vomiting. The first of these is cerebral anemia, which is produced by lowering of blood pressure and slowing of the heart beat, and is relieved when these two phenomena return to normal. The second factor is that of psychic influence, and can be traced to poor appreciation of psychic factors on the part of the surgeon, anesthetist, or anyone who comes in contact with the patient during or just preceding the operation.

The cause of death in spinal anesthesia has received a good deal of attention, and the best evidence points to cerebral anemia rather than respiratory paralysis. Koster and Kosman[3] state that with a marked dilatation of blood vessels within the abdomen and the consequent tremendous fall in blood pressure, the venous return flow to the heart becomes so inadequate that not enough blood can be pumped out to the brain. Failure to supply blood to the brain in sufficient quantity results in a bulbar and cerebral anemia which is responsible for death in spinal anesthesia. When respiratory paralysis ensues, it is consequent to the cerebral anemia. Koster and Kosman[3] have done some very good experimental work to confirm this.

In conclusion, just a word about the duration of anesthesia. The factors which affect the length of anesthesia are the dosage and concentration of the injected drug. The average duration for upper abdominal operations is about one hour, for lower abdominal one and one-fourth to one and one-half hours, while for operations on the perineum and lower extremities many spinal anesthesias will last for one and three-fourths to two hours. There is also a personal variation. Patients having the same operation, and being approximately of the same size, all other things being equal, will often show a variation in duration of anesthesia. However, for all practical purposes the duration is sufficient to perform most major surgical procedures below the diaphragm.

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TREATMENT BY ROENTGEN RAYS IN OVARIAN DYSFUNCTION*

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WITH the rapid extension of radiotherapy to the numerous uses for which it is now recommended, a roentgenologist must be keenly aware of the danger of over-enthusiasm for any particular application because of the danger of later discredit to the entire field of treatment. However, it is equally important, not only for the roentgenologist, but for the surgeon and internist, to keep sufficiently in touch with rapidly developing clinical experience and the accumulating knowledge of the biologic reactions caused by the rays in graded doses on normal and abnormal tissue to give their patients the benefit of such treatment in conditions particularly amenable to it. The profession as a whole, as well as the radiotherapist, should know the relative susceptibility to the rays of the normal tissues of the body and of the different types of malignant growths, and it should know something of the potentialities of treatment by radiation in both acute and chronic inflammatory processes.

A newer field, and one which is in need of critical study, is the use of radiotherapy in certain endocrine dystrophies, particularly because in this field clinical criteria for the diagnosis of disease entities are inexact, and knowledge of the interrelationship of various glands is limited. For the last three years, Dr. Drips and I have been interested in the use of radiotherapy in low dosage in certain cases which may be regarded as representative of the state of ovarian hypofunction or dysfunction. In two previous publications we have considered more fully the basis of diagnosis of this condition and the development of both ovarian and hypophyseal irradiation in its treatment. With only brief recapitulation of the salient features of this syndrome and the technic of the treatment, I wish to present at this time reports of six cases to illustrate some of the possibilities of radiotherapy in low dosage in this field.

Ovarian dysfunction can be rather loosely defined by stating that in a certain group of patients abnormalities of menstruation and of reproduction (sterility or frequent abortion) can, by exclusion of any organic local or general disease or structural abnormality, be attributed only to incompetence of the ovaries themselves. The most common manifestation of this ovarian disturbance is lack of rhythm or cycle in menstruation, exhibiting either suppression of menses (oligomenorrhea or long periods of amenorrhea) or the reverse (too profuse, prolonged, or too frequent flowing). All combinations or interchange of the two phases of alteration are found. Intense dysmenorrhea, distinct from the obstructive type, frequently accompanies these disorders and sterility is likewise an almost invariable corollary.

On general examination, often confirmed by exploratory operation, little change from the normal pelvic conditions can be determined, or at least the variation in size, position, or condition of the uterus and ovaries is within the range of variation found compatible with normal menstruation in many persons.

To limit the group for more accurate study, Dr. Drips and I have endeavored to exclude cases of obvious pluriglandular dystrophy, although in some of these, undoubtedly, the same treatment may have an alleviating effect, and its use may be indicated. That the dysfunction represented is essentially an ovarian hypofunction is evidenced by the palliation afforded in many instances by the use of ovarian extracts and also by the similar symptoms which occasionally arise after pelvic inflammatory disease or after operations in which resection of the ovaries has been performed. Many of the more severe cases have proved intractable to the commercial preparations of ovarian substance now available and

*From the Section on Roentgen-Ray Therapy, The Mayo Clinic, Rochester, Minn. Read before the Association of Resident and Ex-resident Physicians of The Mayo Clinic and The Mayo Foundation, Rochester, Minnesota, October 8 to 10, 1929.

it is in these more resistant cases that treatment by roentgen rays is employed. Two methods of treatment have become fairly well standardized and are in use in many European and in some American clinics; namely, the application, to either the ovaries or to the hypophysis, of a very small amount of roentgen rays. The range of dosage is approximately 5 to 10 per cent of an erythema dose at the depth of the organ treated; rays of short wave length are used. Although we have had occasion to employ both forms of treatment, we have found the ovarian irradiation or a combination of both ovarian and hypophyseal irradiation more effective, as a rule, than the pituitary irradiation alone. Approximately two decades of observation of patients treated by this method, together with many experimental studies and some data based on histologic studies of ovaries from women following irradiation with this dosage, had not shown indication of harmful sequelæ to the patient or to the subsequent offspring. This latter point had been the alarm of the general profession or at least those unacquainted with the significance of dosage of rays in proportion to biologic reaction which can result. Few clinicians hesitate to subject a patient to roentgenographic examination and often repeated examinations of the colon, of the spine and pelvic bones, or of the urinary tract, on any suspicion of disease in these structures, but many of these same clinicians are strongly prejudiced against the use of an almost analogous amount of irradiation in treatment. The recent extensive survey by Murphy of the incidence of congenital or constitutional defects in children born to mothers who had previously been subjected to pelvic irradiation, often in considerable amounts, such as is used in the production of temporary sterility or in the treatment of fibromyomas, should answer conclusively these doubts.

Case 1.—A woman, aged twenty-two years, 5 feet 5.5 inches in height, weighed 170 pounds. Menstruation had begun at the age of thirteen years. It always had been irregular and scanty and had ceased suddenly six years before admission to the clinic. Since then she had menstruated only once and that two years before admission. She had had no vasomotor symptoms and no molimen.

The uterus was small, in the anterior position, movable, and the ovaries were palpable.

Previous treatment had included ovarian extract, 10 grains three times daily, for six months, and intravenous organotherapy, probably with corpus luteum. This treatment had been without results, and no medication had been given for one and a half years.

Irradiation of ovaries was given in June, 1928, and a reduction diet was advised. Supplementary treatment with organ compounds was not given. After irradiation the patient experienced some pain, low in the pelvis, for about a half hour every few days. Six weeks after treatment a dark, hemorrhagic vaginal discharge occurred which continued for five days. The patient returned in August, 1928, encouraged by this indication of return of ovarian function and anxious to continue treatment. Combined pituitary and ovarian irradiation was given. Since then menstruation had occurred every six weeks to the date of the last observation, August 5, 1929. Menstrual flow is normal for one and a half days, and then there is a brownish discharge for three days. The patient experiences slight pelvic discomfort with the periods; her general health is excellent.

Case 2.—A single woman, aged twenty-four years, 5 feet 4.5 inches in height, weighed 240 pounds. Menstruation had begun at the age of eighteen years. It always had been irregular, scanty, and associated with severe pain. There had been excessive gain in weight since puberty. She had frequent headaches.

The uterus was small and lesions were not found in the adnexa. The basal metabolic rate was +12. Obesity was graded 4. There were no acromegalic features.

Previous treatment had included appendectomy and bilateral salpingectomy for early tuberculous salpingitis, and puncture of small cysts in the ovaries, four years before admission to the clinic. Menstruation and general health had not improved following operation.

Irradiation of the ovaries was given in June, 1927, and a reduction diet was advised. Supplementary treatment with organ compounds was not given. Menstruation occurred regularly every month from June, 1927, to May, 1928, at which time septicemia with high fever occurred. Since then there has been no menstruation. During the time of regular menstruation the patient improved greatly in general health and her weight was reduced to 214 pounds.

Case 3.—A married woman, aged thirty-two years, 5 feet, 1.5 inches in height, weighed 213 pounds. Menstruation had been fairly regular until the patient was twenty-two years of age. Since then it had been infrequent and scanty, with intervals from two months to two years, and a rapid gain in weight. Hot flashes were frequent, and molimen was present. The patient desired children. Pseudocyesis occurred twice.

The fundus of the uterus was normal and movable, and lesions were not found in the adnexa. The basal metabolic rate was +15. Roentgenogram of the sella turcica did not show abnormality.

Previous treatment had included ovarian extract over long periods, which occasionally had caused a slight hemorrhagic discharge or increase of molimen.

Irradiation of one ovary was given in January, 1927,

a reduction diet advised, and agomencin (an ovarian preparation) prescribed to be taken for one week before the expected menstruation. Menstrual flow occurred two days after the irradiation and continued for one month, following which normal menstruation occurred from April to August, 1927. Each period lasted about one week. In August the patient became pregnant, and in May, 1928, gave birth to a female child; the child was well developed and apparently vigorous, but died two hours after birth from respiratory failure. The attending physician reported that the birth had been slow and difficult due to the extreme obesity of the mother, and he could account for the death of the child in no other way. Menstruation was reëstablished on one occasion following the birth of the first child before the second pregnancy occurred. A male child weighing 8.5 pounds was born in May, 1929. The child is now five months of age, weighs 16 pounds, and is healthy and well developed in all respects. The mother reports her health better than at any previous time; she mentions particularly that the soreness of the flesh of the entire body, which had been so noticeable during the periods of amenorrhea, is now relieved. Two menstrual periods have occurred since the last childbirth.

This has been the only case in our series in which pregnancy has followed reëstablishment of menstruation. Kaplan has published a series of thirty-eight cases treated for amenorrhea in which menstruation was reëstablished in thirty-two. In thirty of the group who were married and previously sterile, pregnancy occurred in eleven instances, two miscarried, and nine healthy children had been born. Linzenmeier also has reported his experiences, with similar results. Congenital defect attributable to irradiation was not present in any of the children.

Case 4.—A single woman, aged twenty-four years, had begun to menstruate at the age of twelve years. Menstruation always had been irregular, and the average interval between periods was six weeks. Intense dysmenorrhea had occurred for the last nine years. The most severe pain occurred on the first day and always required morphine for relief. The patient occasionally lost consciousness from pain before the hypodermic was given.

There was no pelvic abnormality.

Previous treatment included dilatation and curettage in August, 1925, without relief. Large doses of ovarian extract were taken two weeks before each period, but had given no result after several months' trial. A second dilatation and curettage and insertion of Baldwin tube, which remained in position for six weeks, was likewise without effect.

Irradiation of the pituitary gland was given in June, 1928. There was slight improvement of the period which immediately followed the irradiation. The treatment was repeated in July. The next two periods were much less painful. Because complete relief had not been obtained, an ovarian irradiation was added in September, 1928. Since then the patient has been either free from pain or experiences slight discomfort for a few hours at the beginning of menstruation. This discomfort is amenable to ordinary remedies such as aspirin.

Case 5.—A single woman, aged twenty-four years, had had severe dysmenorrhea for five years following appendectomy. The pain incapacitated the patient for work. Menstruation was irregular, with intervals of five to seven weeks, and there was pain and nausea one week to sixteen days before menstruation began. The patient was confined to bed with each period, vomiting every fifteen to thirty minutes during the first day. There was severe backache and abdominal cramps.

The fundus of the uterus was small and retroverted, and lesions were not found in the adnexa.

Previous treatment included ovarian extract, 10 grains three times daily for two weeks before the menstrual period, corpus luteum, and Hammond's mixture, without effect. Benzol stearate was given to relieve spasmodic pain at the time of the menstrual flow, but could not be retained because of vomiting.

Irradiation of the hypophysis was given in June, 1928. The July period was much better. There was a return of nausea with the next period, although the pain was less severe. A second pituitary irradiation was given in October, 1927. The patient was entirely free from nausea and pain until January, 1928. There was slight dysmenorrhea then, and apprehension of further trouble led to the third treatment. Since this time the patient has been well. She has completed two years of nurse's training since the second treatment, and has not lost any time from duty because of illness.

Case 6.—A single woman, aged thirty years, came to the clinic complaining of menstruation which, although regular, was profuse, and accompanied by intense pain for thirty-six to forty-eight hours. There was a marked state of chronic nervous exhaustion, due chiefly to dysmenorrhea.

The uterus was retroverted and the ovaries distinctly palpable.

Previous treatment included internal shortening of the round ligaments and dilatation and curettage in March, 1929. The ovaries were described by the surgeon as elongated (5 to 6 cm.) with heavy capsules, the surface of which presented rugæ. The cervical canal was not of the rigid type seen in obstructive dysmenorrhea. The menstrual periods in April and May were associated with the same intense pain.

Irradiation of ovaries and of the pituitary gland was given in May, 1929. There was marked relief from dysmenorrhea for the next two periods. A slight return of pain, relatively mild and of short duration, occurred with the third menstruation. The patient gained weight and strength. The amount of menstrual

flow was distinctly lessened. An examination in September, 1929, showed the ovaries still large and tender, and the uterus small and anterior in position. A second ovarian irradiation was given in September, 1929.

Treatment has not been equally successful in all cases; there have been some complete failures, but the results in over 50 per cent of the cases treated have demonstrated conclusively the potential effectiveness of the method. Improvement in results may be expected with more careful study of the types of dysfunction within the group as a whole, and perhaps through combinations of various irradiations of endocrine glands such as those recently advocated by Borak. With the development of more effective extracts of organs, radiotherapy probably will be superseded by direct substitution therapy, but in the meantime it would seem to have a definite field of usefulness.

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CASE REPORT

THE TETANOID SYNDROME IN PREGNANCY

REPORT OF CASE

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In a recent article[1] it was suggested that a group of symptoms, rather commonly observed in pregnant women, probably due to a relative hypocalcemia, be considered as a tetanoid manifestation and that the group of symptoms be classed as the tetanoid syndrome. These symptoms are achy or crampy pains in the legs and thighs, an irritability of disposition unusual to the patient, insomnia, an edema apparently not associated with cardiac or nephritic pathology, and paresthesias of the extremities.

The symptoms comprising the tetanoid syndrome complicating pregnancy are nicely illustrated in the following case report. The patient is one of the series being studied at the Wilder Obstetrical Dispensary through the kindness of Dr. J. L. Rothrock, and I am indebted to the same source for the opportunity to interview the patient after delivery at the Miller Hospital and to study her hospital record.

Mrs. P. F., white, aged 36, gravida X, had one miscarriage at three months between her seventh and eighth pregnancies. Six children are living; three are dead—one due to convulsions at about three months of age, one to meningitis at three months, and one due to a six weeks premature delivery.

The patient herself had scarlet fever at fifteen years and was seriously ill at a hospital for six weeks. Chorea followed this illness and lasted for about four months. At sixteen she had an appendectomy. She has been under observation for tuberculous pleurisy which probably healed. She has also been treated for some tuberculous affection of the eyes. In spite of her illnesses and numerous pregnancies the patient appears strong, healthy and vigorous. Laboratory reports Kolmer Wassermann 4 plus, and Kahn 3 plus.

Her early pregnancies were uneventful. The seventh and eighth, however, presented symptoms identical with the ones here reported, except for the fact that they appeared during the eighth month while with this they appeared, in part, as early as the fourth month.

In this pregnancy edema of the feet and ankles appeared in the fourth month. Towards the latter part of her pregnancy she also had edema of the hands, while that in the lower extremities had become pronounced, pitting deeply on pressure. Varicosities became conspicuous in the later months; considerable areas about the legs and ankles were colored a deep bluish purple over the veins with the adjacent areas a washed-out indigo.

About four weeks before term insomnia became an annoying symptom; the onset of crampy pains in the legs and feet at the same time probably accounts for the insomnia in some degree. She became increasingly nervous and irritable and cried easily. When seen ten days before delivery at the dispensary she impressed one as being mildly hysterical, speaking of her various symptoms with tearful, anxious hopelessness. The urine was free from albumin throughout the pregnancy; B. P. 110/60. Her hands and feet often felt numb. She would pound her hands on her knees in an effort to get some feeling into them.

During the last month she made only one visit to the dispensary because walking was so difficult on account of pain in her feet.

On July 19 she was given 40 units of parathormone intramuscularly. At the same time she began taking calcilact, 1 teaspoonful four times daily. On July 20 the edema had almost disappeared. The paresthesias and crampy pains in the extremities completely disappeared. By the twenty-first the edema was entirely gone, together with most of the discoloration. She slept soundly. On the twenty-second she took a long walk, the first in many weeks. She went into labor on the twenty-second, and spontaneously delivered a baby weighing 8 pounds and 2 ounces after a labor lasting only four hours. This is remarkable in comparison with the two labors preceding, which both lasted five days, with babies weighing respectively 5 pounds 2 ounces and 6 pounds 8 ounces. The puerperium was afebrile throughout. She slept soundly, and was entirely free of nervous manifestations, edema, parasthesias, and cramps.

Discussion.—When one considers the demands made upon the calcium metabolism of the mother by the growing fetus, and by lactation, it is not surprising to find that occasionally the apparatus fails to fully satisfy the demands made upon it. It is an old observation that tetany develops more readily in either humans or animals during pregnancy and lactation, or even during menstruation, than at other times. "Rheumatic contracture of nursing women" was proposed by Trousseau in 1854 as a name for the condition. McCarrison[2] has observed the very marked tendency for the women of Gilgit Valley in India to develop tetany during pregnancy and lactation. Williams[3] gives Dibbelt's estimate of at least 0.17 grams of calcium oxide daily as the average demand for calcium during the second half of pregnancy. Bar (from Williams) calculates that in the last two months this must be increased to 0.638 grams daily.

It is difficult to observe edema in pregnancy without a sense of alarm. Our teaching has been to accept it as a manifestation of toxemia, and toxemia, in turn,

means grave warnings, anxiety, and complete disruption of any ordinary manner of living. Even when the diagnosis of toxemia is made questionable by the absence of the usual accompanying signs, it has been the conservative practice to treat such patients as potential toxemias. When nephritic pathology is absent, however, as in this case, and where other tetanoid symptoms exist, one may conclude that toxemia probably does not exist, nor even threatens; certainly the patient need not be subjected to the discomforts, anxiety—perhaps dangers—which attend anti-toxemia measures. The nature of the edema in this condition is uncertain. In the article referred to, Faltas' conception of it as an angiospasm is given, but this does not seem to be so likely as the idea that it is the result of an alteration in blood-vessel permeability due to the low calcium content of the blood.

Insomnia in these patients is often extremely annoying, not only to the patient herself, but to her physician as well, since she responds so poorly to the usual soporifics. The cause of this symptom is probably related to the other nervous manifestations, including the cramps and paresthesias, and as such would be an expression of the heightened irritability of the nervous system. The bearing of this condition upon the duration of labor has been elaborated somewhat in the reference and is one of its most important aspects. There is a relative inertia of the uterus and a tendency to post-delivery atony. It is in this type of uterus that the muscles may have the power to dilate the cervix, but fail in that expulsive force so imperative to the normal progress of the second stage. Such patients may labor for hours and days, suffering from the pains of labor and despairing because of their futility. The problem is usually solved by some type of operative delivery with a danger all the greater because of the exhaustion of the patient, the probable high level of the fetal head, and the (usually) frequent examinations made to ascertain the cause of the delay. It is remarkable that pituitrin has but little effect upon the uterine contractions in these patients; even extraordinary amounts may be given without perceptibly altering the rate or the force of contractions.

How often this condition may produce a prolongation of labor is not known. An effort is being made at the present time to determine the incidence of the condition in a considerable number of obstetrical patients being observed over a period of one year. Difficult labor due to a discrepancy between the size of the child and that of the birth canal or to unusual positions of the child must of course be ruled out. The chief indication of the condition must be the clinical history—and the condition should be relieved before its possible effect upon the progress of labor becomes apparent.

Treatment.—Measures tending to stimulate calcium metabolism are used, and calcium itself is given in some palatable form. Calcilact has been found an agreeable form to most patients, and four or five teaspoons of this may be given daily. Calcium chloride may be used intravenously, especially in the more severe forms. For stimulating calcium metabolism the parathyroid gland extract used intramuscularly is apparently the most effective agent. Viosterol may be used and seems adequate where the symptoms are comparatively mild.

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218 Lowry Medical Arts Building.

PRESIDENT'S LETTER

THE practicing physician has heretofore looked upon preventive medicine as a state function, to be performed by official boards of health. He has conceived of this function as applying to groups of people and in relation to the discovery and eradication of the causes of such diseases as tend to be endemic, epidemic and pandemic. He appears rarely to have sensed the idea that applied preventive medicine is also an individual matter and is just as surely a part of his business as is the treatment of measles, mumps, whooping cough or diphtheria.

It is certain that this attitude is very largely, if not wholly, responsible for school boards, health boards and lay health associations taking up such work as wholesale vaccinations against certain of the contagions.

The State Board of Health has ever stood ready to aid in making various biologic products for the prevention and cure of disease easily and cheaply available to public and physicians alike. The physician has been quick to avail himself of any such preparations when needed to aid in effecting the CURE of disease, but there he is apt to stop. He has been so ingrained in the habit of trying to cure disease after it has developed that he has been almost stone blind to the necessity of treating the unexposed healthy person for purposes of prevention.

The physician must become health-minded. He must learn to look upon himself as a health physician, *i.e.*, one who is ever on the alert for the recognition of the earliest and hidden signs of disease; one who is ever on the alert to avail himself and his people of the accepted methods for prevention of illness.

The campaign for wiping out diphtheria will be in full swing before long and it will become the duty of each and every one of us to aid earnestly in putting into effect plans for the general use of toxin anti-toxin.

It has been said that if every man, woman and child could be given preventive doses of toxin anti-toxin, diphtheria would be completely eradicated. What a boon that would be! What a boon to childhood! For it is in the tender years that this disease is most contagious, most easily overlooked and therefore most fatal.

The method by which this preventive treatment may be given to large numbers of children will vary in different localities. Whether it shall be done by local doctors, by the health department or by other agency is for the local profession to decide. It would seem that the logical way would be for the local profession to treat those who are not indigent; the rest should be taken care of at public expense.

Before any of this work can be done, however, the public must be apprised of the dangers of diphtheria, preventive value of toxin anti-toxin and its harmlessness.

It is our duty to live up to our ancient traditions. The public looks to us these days to PREVENT disease. They are calling to us. We cannot fail them! The physicians in every locality in the state should take the *initiative* in this work. They can commandeer all the organized health agencies to their service. They can, if they so desire, have the help of the extension service of their Committee of Hospitals and Medical Education, their Committee on Public Health Education, and of any individuals or groups they might consider helpful. They have but to ask to receive.

PHYSICIANS! START THIS CAMPAIGN OF EDUCATION AND CARRY IT TO ITS LOGICAL AND BENEFICENT CONCLUSIONS!

S. H. Boyer

President,
Minnesota State Medical Association.

EDITORIAL

MINNESOTA MEDICINE

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MORE NARCOTIC LEGISLATION

No one is more anxious to prevent narcotic addiction than the physician, and the profession as a whole is only too willing to coöperate with the government regulations necessary to limit the use of habit-forming drugs to proper channels.

Various estimates have placed the number of narcotic addicts in the country at figures ranging from 15,000 to five million. The very nature of drug addiction makes accurate determination of the number of addicts impossible. Even though the low estimate is right every effort should be made to cut it down.

As a result of a seeming increase in the number of addicts in the country the Harrison Act was passed in 1914. Various amendments have been added from time to time and the Porter bills now before Congress have attracted attention to narcotic legislation.

The Harrison Act was supposed to direct the use of narcotics to legitimate channels. What it really was in the main to direct their use *in* legitimate channels. This was so evident that the public gained the impression that the law was directed against the medical profession. By inference at least the medical profession was blamed for a large part of the abuse of a most valuable drug. The fact remains that financial gain on the part of those selling narcotics illegally to addicts is the main cause for the spread of addiction. The percentage of addicts made so by careless physicians must be very small. The few unprincipled physicians who profit by aiding addiction cannot be too severely condemned and should be punished to the full extent of the law. Enforcement of law should be specially directed against such unprincipled offenders.

Certain provisions of the Harrison law have been needlessly strict and irritating to both physicians and pharmacists—so much so that certain pharmacists have refused to handle narcotics at all and certain physicians have refused to treat addicts for fear of penalties attached to infringement of over-rigid and ambiguous regulations. The physician must keep a record of narcotics administered in the office; must use special requisition blanks for morphine tablets; cannot order a prescription containing codein above a certain amount by telephone; must observe certain incomplete regulations in treating an addict in an institution, and so on.

Registration by physicians licensed to administer narcotics is required each year and an oversight does not relieve the physician of the pen-

alty, hich may be $2,000 or imprisonment for five years or both. As a matter of fact, anyone who violates or fails to comply with any provision of the Harrison law is liable to the same penalty.

As though the present law were not stringent enough in regulating physicians, now come the Porter bills mentioned in the editorial of the A. M. A. Journal of February 8, 1930. One of these bills has already been presented to the House and if enacted no physician who has been convicted of any offense against the present narcotic law, no matter how trivial, may ever be licensed to administer a narcotic again. All regulations are to be made by the Commissioner of Prohibition subject to approval of the Secretary of the Treasury. The physician whose rejection of application for a license is impending must show cause why a license should be issued or why his license should not be revoked—in other words is considered guilty until he proves his innocence. The defendant has no voice in determining the place of hearing, whether it be public or private, and no rights as to who shall be subpœnaed as witness. Only the Commissioner of Prohibition may pass on the evidence submitted. Provision is made for appeal to the courts from decisions rendered by the Commissioner.

The second Porter bill provides for the creation of a special Commissioner of Narcotics. As stated in the editorial this centralized bureau in Washington will have dictatorial right in determining what physicians licensed by the respective states shall be allowed to administer narcotics. We physicians will have a Mussolini in Washington to dictate whether or not we may prescribe narcotics. Such high-handed legislation merely sets the scene for trouble, and violations of a needlessly strict law will result.

The present and proposed narcotic legislation is missing the point. Instead of giving dictatorial power to a Bureau in Washington to regulate physicians, it would be far better to concentrate the energy of the Bureau on the illegal peddling of narcotics outside of the profession.

The regulations proposed in these Porter bills border on tyranny, and every physician in the state should do his part in defeating these bills. Write your representative in Washington.

APROPOS OF MEDICAL COSTS

Mark Hanna has been credited with the remark, "Money and machinery entered the world at the same time." He was interested in accumulating and using both—and time is according him much credit. Diagnosticians of social and political ills have not failed of late to feature the load placed on society by the recent triumphal expansion of medical service. Could this have been an isolated expansion while the mass of the people retained their former alignments we would not have so much of the criticism appearing in papers and magazines. A machine age, however, with revolutionary changes in money distribution; high wages, and long periods of unemployment; installment buying and poor family budgeting—these and many more trends have brought not only expensive but chaotic living.

One searches a long article in the American Medical Association Bulletin* for some tangible expression other than excellent historical references and philosophical applications; some intimate criticism of doctors that minimizes their human frailties and points to their group salvation. Doctors are just as much out of social adjustment as other restless groups. We also have been touched by those agencies mentioned by the famous Ohio senator. Who, among us, have bought more cars than doctors, or more radios or cities' "serpents"! The generation before us bought fast horses and profitless farms. We need some of that epicurean advice which "calls not so much for an increase in our estates as a decrease in our desires." A reader chuckles and quotes the Chinese proverb for the benefit of this writer: "The mandarin from his chair sings of the many blessings of poverty!" No medical doctor of today, blessed with even a fair personality, need fear impoverishment. If it is his lot, it may be from accident, but can usually be traced to an overgreediness for fees, an overestimate of his own powers and prerogatives, or an asinine eagerness in investment which sends him "out wool gathering," but returns him "well shorn." Therefore, a lot of this preachment about chang-

*Hall, Harry M., A. M. A. Bulletin, Vol. 24, No. 9, p. 230; "DesCartes Was Right."

The article begins with the quotation, "If ever the human race is raised to its highest practicable level intellectually, morally and physically, the science of medicine will perform that service."—*Rene DesCartes.*

ing the attitude of the public toward us is plain bunk.

Let us adjust ourselves, our attitudes, our fees and hospital costs, to meet fair human needs. Before we launch tumultuous propaganda in order to adjust our patients to our ideas, let us practise a few adjustments among ourselves. It is difficult indeed to keep up with the strides in our own profession; it is necessary to attend many medical meetings and to organize our work at home. No better means of keeping the public with us has ever been devised than to let them know by our work that we are alert and ever seeking the light. Just as we must deal with individuals who need our attention, the public in turn must deal with us largely as individuals.

Medical service must remain a piece of handiwork that machine methods and mass production cannot well do. Louis XIV paid two hundred thousand dollars to the man who dared lance his royal rectum. At the same time, most of his subjects defended their health with charms, grog and prayer. Grog is still considered notably effective by a large percentage of our population. There are obviously gross inequalities in the capacity or ability of people to meet the cost of accident and disease. The one notable equality is that of desire, whether it be in terms of luxuries or necessities. Hence it is that many good folks assume needless burdens of medical and hospital expense, and doctors for the most part find it most difficult to keep them from doing so. For a considerable portion of our economically soundly organized countries a king with empyema gets about the same treatment as does his meanest servant. Indeed, we may answer DesCartes: "Some parts of the world have been remade by doctors."

E. L. T.

CASCARA-AGAR NOT ACCEPTABLE FOR N. N. R.

The Council on Pharmacy and Chemistry reports that, under the name "Cascara-Agar," the Reinschild Chemical Co. markets a preparation stated to contain "15 per cent of a watery percolation of two-year-old cascara bark, which is processed into No. 1 Agar, cut to size" and is recommended for use in constipation. It is stated on the trade package that the preparation is: "A harmless vegetable addition to breakfast food. Each teaspoonful contains a mild and specially prepared solution of Cascara Tea." Since no statement as to the amount of cascara contained in the product was given, the firm was asked to make a plain statement of the constituents of the product. The firm replied giving the method of preparation of the product. However, since no details were given as to the method used to "debitter" the cascara, one cannot say how much of the active principle of cascara was lost in the process of preparation and therefore the amount of cascara in a given quantity of the finished product cannot be judged. Experiments carried out lead to the conclusion that "Cascara-Agar" contains at most only a trace of cascara, and that it is misleading to call the preparation "Cascara-Agar." Information was received that the Reinschild Chemical Co. still markets "Regulin," a product which has been stated to be prepared in the same manner as is "Cascara-Agar." Since the Council does not accept an article under one name if an essentially similar product is marketed by the same firm under another name, this makes "Cascara-Agar" further objectionable. The Council declared "Cascara-Agar" unacceptable because it is an indefinite mixture marketed under a misleading name with unwarranted therapeutic claims, and because an essentially similar product is marketed by the same firm under another name. (Jour. A. M. A., October 26, 1929, p. 1309.)

ANAYODIN NOT ACCEPTABLE FOR N. N. R.

The Council on Pharmacy and Chemistry reports that Anayodin was presented by the Ernst Bischoff Co., Inc., with the statement that it was composed of iodoxyquinolin-sulphonic acid with the addition of 22 per cent sodium bicarbonate, and that from the information before the Council it appeared that Anayodin was a mixture prepared from approximately four parts of 7-iodo-8-hydroxy-quinolin-5-sulphonic acid and one part of sodium bicarbonate, which during recent years has been used in the treatment of amebic dysentery while similar preparations under various trade names had before this been proposed as wound antiseptics. The Council informed the Ernst Bischoff Co., Inc., that, since it was not the discoverer of the preparation, the Council could not recognize a proprietary name for it, but, unless other conflicts appeared, its product would be accepted if marketed as chiniofon, which name the Council had adopted for the mixture represented by "Anayodin," if acceptable tests were provided to insure its purity and uniformity and the advertising revised to meet stated objections. Ernst Bischoff Co., Inc., did not make its preparation acceptable and, accordingly, the Council declared "Anayodin" unacceptable for New and Non-official Remedies because it is an unoriginal preparation, marketed under a non-informing name without an adequate statement of composition; because no evidence was available to show that its identity and uniformity are adequately controlled; and because it is marketed with therapeutic claims which are unwarranted. (Jour. A. M. A., October 5, 1929, p. 1065.)

A PAGE FORUM OF THE COMMITTEE ON PUBLIC HEALTH EDUCATION

How Shall the Doctor Be Paid?

An important current magazine devotes its entire issue to this timely subject and every medical man should find something of interest to him in the discussion. The vexing question of the cost of health is discussed from the angle of the physician, of the economist, the business man, the public health expert and a large number of others who have interesting things to say on the ways and means of extending that commodity as far as possible at a proper cost in the January *Survey Graphic*.

Few of these experts are able to do more than offer pertinent observations. The answer to the problem of medical costs which are at once fair to the purchaser and to the doctor is apparently quite too complicated for any one man's solution. However, here are a few pertinent quotations from these entirely unacrimonious discussions, all of which are illuminating if not conclusive.

"Health is quite as much a social as a personal necessity and if this is admitted there is no dodging the individual and collective privilege and hence duty of attaining as much of it as the medical and social sciences have to offer. Knowledge, indeed, is pooled but its distribution for use is much after the fashion of April showers. It is time the users tapped the pool with a strong sky scraping plan of living." Thus Dr. Haven Emerson, health editor of the *Survey Graphic* and professor of Preventive Medicine, Columbia University, in a somewhat figurative flight which serves as introduction to the survey.

"If, as is generally supposed, doctors' fees are a preponderating item in making adequate care impossible," says Dr. George Edward Follansbee, chairman of the Judicial Committee, American Medical Association, "it would follow that a vast sum is flowing into the pockets of the medical profession. . . . Each doctor knows comparatively and probably closely the incomes of his fellow practitioners. His guess is that the income of a large portion of the profession will average in the neighborhood of $5,000 to $6,000 a year. An income of $5,000 to $6,000 a year does not appear to be excessive for a man who has spent his life up to 28 years of age in study and preparation and some $12,000 in expense before he begins earning."

Walton H. Hamilton of the Yale Law faculty, writing as economist, patient and parent, takes up the discussion at this point. "The technology of medicine has advanced," he remarks. "Its organization has lagged. . . . The problem of mending our arrangements so that the ordinary man has a real chance at adequate service and at a tolerable cost needs to be attacked in the way disease is attacked. The right answer is probably that there is no right answer. In the spirit of constructive inquiry we can only seek the rightest answer we can find."

"Only the well-to-do and the dependent in Columbus [Ohio] have the medical care at child-birth approximating modern standards" is the conclusion of Mary Louise Mark of the Ohio State University faculty who inquired into and analyzed the costs and service to 540 "run-of-the-mill" babies in Columbus. "The very poor and the lower range of those classified as intermediate have far from satisfactory care. The more intelligent of the parents in close circumstances are the chief sufferers because they are conscious of the risks involved for both mothers and babies."

"Any business which is organized in such a way as to discourage the masses from becoming customers is not on a solid economic basis no matter how excellent its scientific attainment may be," observes Edward A. Filene, who, as president of William Filene's Sons Company of Boston, has brilliantly demonstrated his knowledge of successful business.

"The health department can help the doctor increase his legitimate private practice through its program in diphtheria immunization, for instance," declares Dr. Shirley W. Wynne, New York City's Commissioner of Health. "But the doctor must remember that once a health department embarks upon a program to encourage people to go to a private practitioner he must do his part to keep that patient by his professional competence, moderate charges and effective follow-up work."

The industrial hospital, the pay clinic, certain Canadian experiments in sickness insurance and outlines of the work of the foundations to fight disease also occupy pages of the survey.

REPORTS AND ANNOUNCEMENTS OF SOCIETIES

MEDICAL BROADCAST FOR THE MONTH

The Minnesota State Medical Association Morning Health Service

The Minnesota State Medical Association broadcasts weekly at 10:15 o'clock every Wednesday morning over Station WCCO, Minneapolis and St. Paul (810 kilocycles or 370.2 meters).

Speaker: William A. O'Brien, M.D., Associate Professor of Pathology and Preventive Medicine, Medical School, University of Minnesota.

The program for the month of March will be as follows:

March 5—"High Blood Pressure"
March 12—"Self Medication"
March 19—"Bronchitis"
March 26—"Paralysis of Face"

MINNESOTA STATE MEDICAL ASSOCIATION

Plans for the program of the annual meeting of the Minnesota State Medical Association to be held in Duluth July 14, 15 and 16, are well under way.

The symposia on traumatic surgery, immunology, cancer, rheumatic fever, anesthesia and therapy suggested as program headliners by the program committee have been approved by a majority of association members. The interesting and important aspects of each are accordingly being apportioned to the three days' sessions with the most distinguished men available to lead discussion and demonstration. Among the latter is Dr. Homer F. Swift, New York, who has been asked to discuss rheumatic fever.

Interesting and unusual entertainment is also being arranged for wives of members who attend.

UNITED STATES PHARMACOPŒAL CONVENTION

The eleventh decennial United States Pharmacopœal Convention will convene in Washington, D. C., May 13, 1930. Eligible organizations and colleges should obtain credential blanks from the Secretary of the Convention, Lyman F. Kebler, M.D., 1322 Park Road, N. W., Washington, D. C. Organizations entitled to representation at the convention include Medical Schools, State Medical Associations, and the American Medical Association.

UPPER MISSISSIPPI MEDICAL SOCIETY

The annual meeting of Upper Mississippi Medical Society was held Jan. 30, 1930, at Brainerd, Minnesota, Dr. T. C. Davis presiding. Minutes of the previous meeting were read and approved.

The annual financial statement showing a certificate of deposit of $446.49 and balance in checking account of $493.36—total cash on hand $939.85—was read and accepted.

The Board of Censors reported favorably on the application of Dr. Knute Holmgren, Upsala, Minn. It was moved, seconded and carried that the rules be suspended and the secretary cast the unanimous ballot for Dr. Holmgren, who was thereby declared duly elected to membership in the Upper Mississippi Medical Society.

Meeting places were selected for the year 1930 as follows: Spring meeting, Wadena; summer meeting, International Falls, preferaby during the month of July; fall meeting to be arranged if possible in joint session with the Stearns-Benton County Society, the place of meeting and time to be decided by the presidents and secretaries of the two societies.

Officers elected for the ensuing year are: President, Dr. G. I. Badeaux, Brainerd; First Vice-president, Dr. W. A. Miller, New York Mills; Second Vice-president, Dr. A. F. Gaalaas, Wadena; Secretary, Dr. F. F. Kumm, Wadena. Dr. J. A. Thabes and Dr. J. S. Grogan were elected delegates to the state meeting, with Dr. B. W. Kelly and Dr. F. F. Kumm as alternates. Dr. J. A. Thabes was re-elected censor for a period of three years.

The following resolution was adopted: Inasmuch as it is reported that there is at the present time in some parts of the State a desire to reduce the State dues, be it hereby resolved that it is the sentiment of the Upper Mississippi Medical Society that the State Association has done efficient work in the past few years and that this has been made possible by the increased dues; that any reduction in dues would be detrimental to the efficiency of the Society and that our delegates be so instructed and that a copy of this resolution be sent to the State Secretary.

The scientific program included the following presentations:

Treatment of Vomiting of Pregnancy
Dr. Milton G. Brown, Brainerd

Present Method of Treatment of Tuberculosis
Dr. F. F. Kumm, Supt. Fair Oaks San., Wadena

An Informal Surgical Discussion with Lantern Slides
Dr. Harry Ritchie, St. Paul

The Effects of Sun in the Prevention and Treatment of Disease
Dr. W. R. Ramsey, St. Paul

Discussion of State Problems
Dr. S. H. Boyer, President of Minnesota State Medical Society, Duluth

Moving Pictures
Surgical Treatment of Peptic Ulcers
(Davis & Geck)

Resolutions thanking the doctors appearing on the program were passed by the society.

The meeting adjourned.

G. I. BADEAUX,
Secretary.

RICE COUNTY MEDICAL SOCIETY

A meeting of the Rice County Medical Society in conjunction with the Auxiliary, Nurses and Doctors' wives, was held February 6 at the State School for the Feeble Minded at Faribault. Dinner was served at 6:30 p. m. in the Main building under the auspices of Dr. and Mrs. Murdoch.

Dr. George Earl, Saint Paul, presented two subjects: (1) "Surgical Indications (non-thoracic) in the Minimal Tuberculosis Patients" and (2) "Economic Changes in the Practice of Medicine." Response on the part of members was led by Dr. W. D. Beadie, superintendent, Cannon Falls Sanitarium, and Dr. D. E. McBroom, superintendent at Cambridge.

Dr. W. E. Wilson of Northfield expressed appreciation and thanks to Dr. and Mrs. Murdoch for their generous hospitality in behalf of the society. Dr. Murdoch in turn expressed hearty appreciation to Dr. Earl for his instructive and interesting talk.

Mrs. Adolph M. Hanson, in the absence of Dr. Hanson, expressed gratitude and appreciation for the friendly attitude and assistance accorded Dr. Hanson in his research work on Karkinolysin for the treatment of malignancy, by the members of Rice County Medical Society and others.

Mrs. M. L. Mayland, Secretary of the Auxiliary, briefly outlined the aims and objects of her society.

Dr. Norman Lende, Faribault, was unanimously elected to membership.

C. J. Plonske,
Secretary.

OF GENERAL INTEREST

Dr. Clyde H. Fredrickson, formerly of the Mayo Clinic, Rochester, where he held a surgical fellowship, is now associated with the Great Falls Clinic, Great Falls, Montana.

Dr. Mary C. Ghostley of International Falls will act as superintendent of the Lake Julia Tuberculosis Sanatorium, Puposky, Minnesota, in the absence of Dr. R. L. Laney, who is taking a year's vacation.

Dr. Frederick W. Schlutz of Minneapolis, for sixteen years associated with the University of Minnesota Medical School and since 1924 Professor of Pediatrics, will move to Chicago about April 1st to become Professor of Pediatrics at the newly formed Department of Pediatrics at the University of Chicago.

The Secretary of the Minnesota State Medical Association announces that the American Commission of the International Hygiene Congresses invites us to send a representative to the word health sessions to be held in Dresden May 15 to September 30, 1930. If there is anyone expecting to be in Dresden, or if there is anyone who wishes to make the trip, which will cost $774, application should be sent to Dr. E. A. Meyerding, 11 West Summit Avenue, Saint Paul.

Dr. Leland S. Fuller of Chicago has joined the Morsman Eye, Ear, Nose and Throat Clinic of Hibbing and Virginia. Dr. Fuller comes from Cook County Hospital, where he has recently completed a service in the eye, ear, nose and throat department. He is a graduate of Rush Medical College. The personnel of the Morsman Clinic is now as follows: L. W. Morsman, M.D., Fred McK. Ruby, M.D., Andrew Sinamark, M.D., and L. S. Fuller, M.D.

A gold medal will be given to the best scientific exhibit submitted by any single individual at the Annual Meeting of the Minnesota State Medical Association in Duluth, July 14, 15 and 16.

This is the first award of the sort ever given. Its object is to encourage private participation in exhibits and it is the gift of the Southern Minnesota Medical Association, which proposes to use the surplus in its treasury for the purpose.

Dr. Owen Wangensteen was appointed chief of the Department of Surgery at the University of Minnesota Medical School, January 1, 1930. Dr. Wangensteen received his B.A. degree in 1919 and his M.D. in 1922. In 1925 he received his Ph.D. degree after three years' work in Medicine on the campus and at Rochester, Minnesota. Upon the resignation of Dr. Arthur Strachauer as chief of the surgical department in 1925, Dr. Wangensteen was selected to succeed Dr. Strachauer and availed himself of the opportunity to pursue special studies here and abroad in preparation for his present position. He studied in Europe between August, 1927, and September, 1928, under Dr. Fritz de Quervain at Berne, Switzerland, and in the physiological laboratory there, under Dr. Leon Asher, in addition to visiting numerous medical centers in Europe.

Sixty officers, chairmen and committee members of the Minnesota State Medical Association attended a meeting called by President S. H. Boyer for the purpose of coördinating association activities at the Leamington Hotel, Minneapolis, February 10.

"Attendance and interest displayed at this meeting were more than satisfactory," Dr. Boyer declared. It was the first meeting of the sort in the history of the Association.

A plan for an extension course particularly impressed the meeting. It is to include evening programs of the Minnesota Public Health Association; serum and vaccine therapy, heart disease, the State Board of Health, subjects of scientific interest, such as dermatology and lectures, to be provided by the Extension Division of the University of Minnesota.

A similar meeting of the chairmen will be called by Dr. Boyer this spring.

NEW AND NON-OFFICIAL REMEDIES

The following articles have been accepted by the Council on Pharmacy and Chemistry.

ABBOTT LABORATORIES, INC.

Butesin Picrate Eye Ointment

LAKESIDE LABORATORIES, INC.

Ampoules Dextrose (d-Glucose) 10 Gm., 20 cc.

Ampoule No. 51 Sodium Cacodylate 0.243 Gm. (3¾ grains), 5 c.c.

H. K. MULFORD CO.

Pneumococcus Antibody Solution, Types I, II and III Combined-Mulford, four 50 c.c. double-ended vials.

The following articles have been exempted and included with the List of Exempted Medicinal Articles (New and Non-official Remedies, 1929, p. 481):

DAVIES, ROSE & CO., LTD.

Pil. Digitalis (Davies, Rose)

KINGS COUNTY PACKING CO.

Sac-A-Rin Brand California Bartlett Pears

Sac-A-Rin Brand California Tidbits Hawaiian Pineapple

Sac-A-Rin Brand California Royal Anne Cherries

LAKESIDE LABORATORIES, INC.

Ampoule No. 64 Calcium Chloride 10%

TRUTH ABOUT MEDICINES

Diphtheria Toxoid-National.—A diphtheria toxoid (New and Non-official Remedies, 1929, p. 368) prepared from seven-day cultures of the diphtheria bacillus that yield a toxin having an L+ dose of not less than 0.25 c.c. The toxin is treated with formaldehyde. The finished product is tested for antigenic potency. The product is marketed in packages of three vials (one immunization treatment); in packages of one vial (fifteen immunization treatments); packages of forty-five vials (fifteen immunization treatments). The National Drug Co., Philadelphia.

Scarlet Fever Streptococcus Antitoxin-Cutter.—A scarlet fever streptococcus antitoxin (New and Non-official Remedies, 1929, p. 350) prepared by the method of Drs. Dick by license of the Scarlet Fever Committee, Inc. It is marketed in packages of one syringe containing 2,000 units, and in packages of one syringe containing 6,000 units. Cutter Laboratory, Berkeley, Calif.

Typho-Bacterin Mixed (Triple Vaccine TAB).—This product (New and Non-official Remedies, 1929, p. 380) is also marketed in packages of thirty 1 c.c. vials, being ten immunizations of three doses each. H. K. Mulford Co., Philadelphia. (Jour. A. M. A., January 4, 1930, p. 31.)

Ampules Sodium Cacodylate-Mulford, ¾ grain, 1 c.c.—Each ampule contains sodium carcodylate (New and Non-official Remedies, 1929, p. 73) 0.05 Gm. (¾ grain) in c.c. of sterile solution, with 1 per cent of benzyl alcohol. H. K. Mulford Co., Philadelphia.

Ampules Sodium Cacodylate-Mulford, 3 grains, 1 c.c.—Each ampule contains sodium cacodylate (New and Non-official Remedies, 1929, p. 73) 0.2 Gm. (3 grains) in 1 c.c. of sterile solution, with 1 per cent of benzyl alcohol. H. K. Mulford Co., Philadelphia.

Ampules Sodium Cacodylate-Mulford, 5 grains, 1 c.c.—Each ampule contains sodium cacodylate (New and Non-official Remedies, 1929, p. 73) 0.32 Gm. (5 grains) in 1 c.c. of sterile solution, with 1 per cent of benzyl alcohol. H. K. Mulford Co., Philadelphia.

Erysipelas Streptococcus Antitoxin (Concentrated)-Mulford.—This product (New and Non-official Remedies, 1929, p. 349) is also marketed in packages of one 10 c.c. syringe containing 500,000 protective units. H. K. Mulford Co., Philadelphia. (Jour. A. M. A., January 11, 1930, p. 105.)

Curdolac Casein-Bran Improved Flour.—A flour prepared from casein, carbohydrate-free bran, and soya bean, to which leavening and flavoring have been added. It may be used for the preparation of muffins or bread having a comparatively low carbohydrate content and low food value, with bulk. Curdolac Food Co., Waukesha, Wis.

Curdolac Soya-Bran Flour.—A flour prepared from soya bean and a starch-free bran with a leavening mixture. It may be used for the preparation of bread and muffins for use in diets in which a comparatively low carbohydrate content is desired. Curdolac Food Co., Waukesha, Wis.

Curdolac Breakfast Cereal.—A medicinal food prepared from soya beans blended with wheat products, including starch-free bran. It may be used as a hot food in diets in which a comparatively low carbohydrate content is desired. Curdolac Food Co., Waukesha, Wis.

Curdolac Casein Compound.—A flour prepared from casein, vegetable fiber and a leavening mixture to which sodium chloride and gluside are added. It may be used for the preparation of carbohydrate-free bread, muffins, cake, etc., for use in diets in which a relatively low carbohydrate content is desired. Curdolac Food Co., Waukesha, Wis.

Curdolac Soya Flour.—A flour prepared from the soya bean. It may be used for the preparation of foods in diets in which a relatively low carbohydrate content is desired. Curdolac Food Co., Waukesha, Wis.

Curdolac Wheat-Soya Flour.—A flour prepared from soya beans, starch-free bran and a small proportion of wheat, with leavening and flavoring. It may be used for the preparation of muffins, cakes, waffles, etc., of well balanced food value for use in restricted diets. Curdolac Food Co., Waukesha, Wis.

Curdolac Soya-Cereal Johnny Cake Flour.—A flour prepared from soya beans and cereal products to which leavening and flavoring have been added. It may be used in the preparation of muffins, cakes, waffles, etc., for use in diets relatively low in carbohydrate, designed for those who cannot use products made with bran. Curdolac Food Co., Waukesha, Wis.

Curdolac Soya-Bran Breakfast Food.—A medicinal food prepared from soya beans and a starch-free bran, to which has been added leavening, flavoring, gluside, and oils without food value. It may be used in diets in which a low carbohydrate content is desired. Curdolac Food Co., Waukesha, Wis. (Jour. A. M. A., January 18, 1930, p. 185.)

CONSULTATION BUREAU

WM. A. O'BRIEN, M.D., *Director*
Minnesota State Medical Association
11 West Summit Avenue
Saint Paul, Minnesota

1. *Question.*—I have a patient, forty-two years old, with a lump in the breast. I wish to know how to make a biopsy, and what the diagnostic possibilities are.

Answer.—The indications for a biopsy of a lump in the breast in a woman past thirty years of age are very definite. While attachment to the skin with dimpling and retraction of the nipple are suggestive of malignancy, no absolute diagnosis can be made without removing the entire mass. Many malignant tumors are not attached to the skin nor do they show retraction of the nipple. While the consistence of the mass on palpation is helpful, very hard tumors are sometimes proven to be benign on biopsy. The presence or absence of enlargement of the lymph nodes in the axilla is not helpful because they may be enlarged from another cause.

The ideal method for preparation for a biopsy is to get the patient ready for a radical resection of the entire breast and axillary contents. The skin should be prepared in the usual way, and the axilla shaved. Many surgeons use local anesthesia to remove lumps which they believe to be benign, so that if there is any delay in getting the report no time is lost with a general anesthesia. We will assume that the lump can be removed under local anesthesia. Infiltration with novocain can be done, and the entire mass excised. In no instance should the lump be cut into before removal. After the mass and surrounding tissue have been removed, it is advisable for the surgeon to make an incision through the tumor. In 95 per cent of the cases, a diagnosis can be made on the gross appearance. If the tumor is very hard and shows no definite border, it is probably malignant. If in addition, the surface is concave and there are multiple yellowish points present, it is malignant. In only 5 per cent of the cases is it necessary to resort to the frozen section. Many mistakes are made on rapidly made frozen sections, and it has been found advisable to rely on the gross appearance. If the mass is a fibroadenoma, it may be recognized by the sharp border. A definite capsule is present. When a fibroadenoma is incised, the edges bulge because the tumor is growing under pressure in a limited area. The surface of fibroadenomata varies from hard structures to the soft appearance of a rapidly growing tumor. Rapidly growing tumors are often found in conjunction with pregnancy or lactation. Cystic disease has a variable appearance. Sometimes there is nothing more than a diffuse induration which is not hard in which may be found either small or large cysts. Sometimes these masses prove to be a single cyst filled with fluid under pressure. Traumatic fat necrosis also gives a similar appearance to malignancy grossly, but the characteristic signs of a malignant tumor are absent. On section, fat necrosis shows definite changes which are very characteristic. If the mass is due to tuberculosis, a similar change, characteristic of the disease, will be found in the frozen section.

When a diagnosis of carcinoma is made, there is only one treatment to be done, providing metastases have not been demonstrated elsewhere before the biopsy is made. It is always wise to search the supraclavicular space and to do an *x*-ray of the chest in suspected cases. Only radical breast amputations should be done if the tumor has not extended beyond the breast and axillary contents. There is no known way of making an accurate clinical diagnosis. The practice of waiting to see what will happen to a lump in the breast is very bad. Many women have lost their only chance by local treatment.

2. *Question.*—Please give me the main points in the diagnosis and treatment of gas gangrene.

Answer.—Gas gangrene is a condition found in certain kinds of wounds, and is characterized by rapid spread, extensive destruction of tissue, foul discharge, and the formation of gas in the tissues. It always occurs secondarily to a wound. The odor is of a very peculiar, pungent quality, and once smelled is never forgotten. A characteristic finding is a sensation of crepitus in the tissue. The extent of the process can also be confirmed by an *x*-ray of the affected part. When opened, the muscles show a definite, dark, reddish brown discoloration and do not contract when stimulated. From 80 to 90 per cent of all cases are due to the bacillus welchii.

An antitoxin has been known since 1917. It is an antitoxic serum prepared from the blood plasma of horses, highly immunized against the toxin of bacillus perfringens or bacillus welchii. It may be purchased under the name perfringens antitoxin. For prophylaxis, 25 units are used. For treatment, an initial dose varies from 40 to 100 units, given intramuscularly followed by 40 units intravenously. From 40 to 80 units intramuscularly are given each day thereafter as indicated. The bacillus welchii is found in the intestinal tract of nearly all adults and about 40 per cent infants under one year of age. It forms spores under certain conditions and is liable to live in soil for a considerable time. It is possible to develop human infections from soil contaminated with human excreta. After the antitoxin is given, it is advisable to trim away all necrotic tissue and irrigate the wound with Dakin's solution. The use of hydrogen peroxide packs and irrigations is also recommended. If an extremity is involved, amputation is usually done. Gas gangrene, if it is not treated, practically always terminates fatally in from forty-eight to seventy-two hours. The condition must be recognized early and the proper treatment done at once. Favorable statistics were compiled during the war, and the evidence in favor of serum has grown since. The antitoxin may be purchased in three strengths, which includes the double strength and the concentrated preparation. Tetanus must also be kept in mind with these wounds, and many investigators use both sera in badly infected cases.

OBITUARY

Louis M. Benepe
(1862-1930)

Dr. Louis M. Benepe of Saint Paul died at his home February 14, 1930, after an illness of five months. He was 67 years of age and had practiced in Saint Paul for forty-two years.

Dr. Benepe was born in Columbia, Missouri, October 27, 1862. He was a graduate of Valparaiso University in Valparaiso, Indiana, and obtained his medical degree at Missouri Medical College in Saint Louis. He came to Saint Paul in 1888.

Dr. Benepe had been a member of the Ramsey County Medical Society since 1891, and had also been on the medical staffs of the Ancker, St. Joseph's and St. Luke's hospitals. He was also a member of the Triune Lodge, Knights of Pythias, and of the Damascus Commandery, Osman Temple of the Shrine.

Dr. Benepe is survived by his widow, Mrs. Leila A. Benepe; five daughters, Ruth and Alice Benepe, Mrs. Helen Vandersloot, Mrs. D. L. Cummings, all of Saint Paul, and Mrs. Frank Munoz of Wichita, Kansas; three sons, Dr. Louis M. Benepe, Jr., of Saint Paul, Dr. James L. Benepe of Dent, Minnesota, and R. S. Benepe of New York City. One brother, Dr. John Benepe of Anderson, Indiana, also survives him.

Paul Burns Cook
(1877-1930)

Dr. Paul B. Cook, well known specialist in urology and dermatology, of Saint Paul, died February 9, 1930, at Miami Beach, Florida. Dr. Cook had practiced in Saint Paul for over twenty-five years and since 1919 had been associated in the practice of his specialty with Dr. Charles Freeman.

Born in Rochester, Minnesota, the son of George Clinton and Elizabeth Burns Cook, on February 17, 1877, Dr. Cook obtained his M.D. degree at the University of Minnesota in 1900. He joined the Ramsey County Medical Society in 1902, was vice president of the Society in 1906, and in 1907 spent a year studying abroad, spending most of the time in Vienna. In 1911 and 1912 he acted as Assistant Health Commissioner and instituted valuable methods in the administration of the office.

Dr. Cook was one of the first medical men in Saint Paul to enlist, and after preliminary training at Fort Riley and serving at Camp Funston, he went abroad as Major in the 88th Division. Fond of travel, the foreign shores visited by Dr. Cook included Hawaii, another trip to Vienna in 1917, a world tour two years ago and last year a vacation spent in Africa.

Besides being a member of the Ramsey County Medical Society, Dr. Cook was also a member of the Minnesota Academy of Medicine and the Minnesota State Urological Society. He also belonged to the Town and Country Club, Somerset Golf Club, the Minnesota and University Clubs.

Dr. Cook is survived by two sisters, Mrs. Frank B. Kellogg and Mrs. Frank J. Ottis of Saint Paul, and a brother, George Cook of Rochester.

Henry F. Hoyt
(1854-1930)

Dr. Henry F. Hoyt, formerly a resident of Saint Paul but for the past twenty years living at Long Beach, California, died at Yokohama, Jan. 21, 1930, while en route from Manila.

Dr. Hoyt's long life of various activities has been depicted in his autobiography published last summer under the title "A Frontier Doctor," the introduction of which was written by former Secretary of State Frank B. Kellogg.

Dr. Hoyt's parents came to Saint Paul in 1848 and settled on a farm near Snelling and Larpenteur Avenues. Here the doctor was born Jan. 30, 1854.

At the age of eighteen Dr. Hoyt accompanied the expedition that established the boundary line between the United States and Canada from Glyndon, Minnesota, to Pembina, North Dakota. Returning to Saint Paul a year later he took up the study of medicine in the office of Dr. J. H. Murphy and in 1875 was placed in charge of the Church Hospital, now St. Luke's. He later entered Rush Medical College, where he received his degree.

Upon graduation Dr. Hoyt began practice in Deadwood, South Dakota. According to his own story undertakers were more needed there than doctors so he journeyed to the Texas Panhandle country on the strength of a report that an epidemic of smallpox was raging there. For the next twenty years he was, in turn, doctor, cowboy, miner, vigilante and doctor again. In Texas he became well known as a sprinter and boxer. From 1893 to 1898 he was chief surgeon for the Great Northern railway.

When the Spanish-American war broke out Dr. Hoyt entered the service and eventually became the ranking Army surgeon in the Philippines under General Frederick D. Grant. In this capacity he was in twenty-five engagements and at Marilao, Luzon, received two flesh wounds but stayed on duty. For this he was cited for bravery twenty-five years later.

Retiring to Saint Paul after the war, Dr. Hoyt became city health commissioner. Last fall, he and a sister, Mrs. George Wilson, of Minneapolis, sailed for the Philippines in order to spend Christmas with a sister, Mrs. George Harvey, of Manila. On board the ship Dr. Hoyt had the unique experience of hearing a review of his own book, "A Frontier Doctor," broadcast by radio from San Francisco.

Besides his sisters, Dr. Hoyt is survived by one son, John Terrell Hoyt, and a stepson, Owen H. Gray.

PROCEEDINGS OF THE MINNESOTA ACADEMY OF MEDICINE

Meeting of January 8, 1930

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, January 8, 1930. Dinner was served at 7 p. m. and the meeting was called to order at 8 p. m. by the Vice-president, Dr. J. S. Gilfillan, of St. Paul. There were 32 members present.

Minutes of the December meeting were read and approved.

The Secretary read a letter from Dr. Gustav Schwyzer of Minneapolis, resigning as President of the Academy. A motion was carried that Dr. Schwyzer's resignation be accepted and the Academy proceed to ballot for another president. Dr. Emil Geist, Minneapolis, was unanimously elected President for the year 1930.

Dr. Arnold Schwyzer (St. Paul) reported the following case* of Endothelioma of the Pleura removed by operation. Specimen was shown.

This morning we had an unusual operation on a young man, 23 years old, who had noticed a persistent pain in the right side of his chest for two years. *X*-rays made several times during the last year showed a tumor originally the size of a large plum gradually growing larger, apparently in the periphery of the lung. A bulging was noticed in November and biopsy showed endothelioma. First we resected a rib above and one below the bulging area, that is, about 12 cm. of the sixth and eight ribs. It was done under moderate over-pressure. The tumor was adherent to the diaphragm. This area of the diaphragm was excised, and a tight closure of the wound was made. This evening, 8 hours after the operation, the patient is reported doing well.

Neither Sauerbruch nor Lilienthal reports an operation for endothelioma of the pleura in their books on surgery of the chest.

(Addendum: Recovery was very smooth. The patient was out of bed four days after the operation and left the hospital one week after operation, feeling well.)

DISCUSSION

Dr. E. T. Bell (Minneapolis): This is a most unusual tumor and we have never had anything like it in our experience in the laboratory. The biopsy shows it pretty clearly to be an endothelioma. It is a cystic circumscribed tumor attached to the pleura over the ribs. The operation was much simpler than had been anticipated. It is the only case on record, I think, of an endothelioma that has been operated upon successfully. I am inclined to think it will be cured because it does not seem to involve any surrounding tissues.

Dr. J. S. Gilfillan (St. Paul): About 25 years ago I had a case come to me with a tumor just over the heart. We did not have *x*-ray then and we did not have available any pressure apparatus. At first I thought that in order to prevent the collapse of the lung I might pass large needles with sutures and get at the lung and hold it, but I did not do that. I opened the chest and found the tumor had involved the left lung. I excised four ribs with the tumor and lifted the lobe of the lung out and excised about one-half of the lung. I rotated the remaining portion of the lung and sewed it around the opening and closed the opening. There was apparently no shock from the operation and the patient felt fine the next day. That was an endothelioma; it was wider and flatter than Dr. Schwyzer's. About a year later I saw the patient, and at that time he had metastases in the vertebræ.

Dr. Herbert Jones (Minneapolis): About two years ago I had a man come to see me complaining of pain in his right shoulder. I went over him for possible trouble in the cervical nerves, a lung condition, or aneurysm, and they seemed to be all negative. I could not make a diagnosis. He did not seem to be getting any results and left me and went to one of the clinics and they *x*-rayed him and found a pleural effusion. They tapped him, and after that his pain was much worse. He then went to an osteopath and later came back to me, this last fall. He died October 30, 1929. When he returned to me he presented the appearance of a large tumor of the chest, but he was in such bad shape that I did not do anything but try and keep him comfortable. He had had one son die of Hodgkin's disease, and a daughter has tuberculosis. I will read parts of the pathological report from the University of Minnesota:

"The right diaphragm is very firm and rigid. The left pleural cavity contains 200 c.c. of straw-colored fluid. The right cavity is obliterated by firm white tumor tissue except for two pockets of straw-colored fluid of about 200 c.c. each, which are located posteriorly. The pericardial sac contains about 20 c.c. of slightly cloudy fluid. The right side of the sac is injected and roughened from extension of the tumor tissue. The right lung is firmly adherent to the chest wall, the diaphragm and the mediastinum by a firm white growth of tumor tissue up to 3.5 cm. in thickness. This growth has gone through the right diaphragm and is adherent to the liver. The nodes of the bronchi are normal to inspection.

"The spleen weighs 90 grams. The diaphragmatic surface of the spleen is firmly adherent to the diaphragm by a small firm white growth 3 cm. across. On section there are many small healed miliary tubercles.

"The liver weighs 1,100 grams. It shows moderate evidence of chronic passive congestion and is firmly adherent to the diaphragm by firm white tumor tissue. The gallbladder contains two small stones.

"Diagnosis: Endothelioma of the pleura with extension to liver, pericardium, lung, diaphragm and spleen."

I was absolutely unable to make a diagnosis in that case, and no opportunity for biopsy presented itself as in Dr. Schwyzer's case. Although I have had autopsies of a large per cent of my cases, I have never before had a case of endothelioma of the pleura and do not see how I could diagnose another.

*Case to be published in full at later date.

DR. HILDING BERGLUND (Minneapolis): A moment's consideration of the diagnosis might be worth while. Could the diagnosis have been made a year ago? I believe not. A circumscribed endothelioma of this configuration is such a rarity that the possibility would certainly not be considered without biopsy. The mass was too well circumscribed and rounded to fit in with a carcinoma of the lung of the solid block type. The picture could just as well have indicated a cyst or an encapsulated empyema. I believe a needle was inserted in the mass about a year ago and about 10 c.c. of clear fluid, not further described, were obtained. With nothing but these facts to go by, I do not believe any very aggressive action was indicated at that time. Now the diagnosis was made possible only through the bulging of the tumor between the ribs and the result of the biopsy was a surprise. It is gratifying to think that the patient was no worse off today than he would have been if it had been possible to decide about an operation a year ago.

One diagnostic procedure, which often proves of value in cases of this type, should be mentioned: a pneumothorax performed before an operation. With the collapse of the lung new possibilities offer themselves for *x*-ray differentiation between intra- and extrapulmonary tumors. Adhesions in one direction or other likewise present themselves. Jacobaeus and Key used to be very fond of this procedure before operation, but of course it is not always necessary.

DR. SCHWYZER (St. Paul): If we had made a preliminary diagnostic pneumothorax we would have had the lung fall away entirely from the growth and we could then have gone in with more assurance of success. However, even if otherwise, it would not have changed our decision to explore; and as a part was adherent to the diaphragm by soft friable neoplastic tissue, it would probably have been torn off and given a chance for dissemination in the pleura.

Dr. Gilfillan's case is the only one that I have heard of that was operated on. I looked through several books rather hurriedly this afternoon and could not find a case.

I forgot to mention the fact that a year ago there was some fluid taken away from this growth.

The sensible thing to do, after the wound is healed, will be raying the area. We shall probably use radium, very strongly filtered so that the skin will not be damaged. I have a case in the hospital now who, a year ago, had had a little over 7,000 mg. hours in one session after an operation of an acousticus tumor in the cerebellopontine angle. No hair fell out after that application. She is in the hospital again now and getting this time 6,000 mg. hours.

DR. ZIMMERMANN (St. Paul): I would like to ask Dr. Schwyzer how he could do this without positive pressure?

DR. SCHWYZER: We used positive pressure. It was done in the simplest way. The patient was getting oxygen all the time through tubes put into the nose, with about 20 cm. water pressure. The patient did not have his mouth closed; there was just a little extra oxygen and a little extra pressure. I could have steadied the mediastinum by getting hold of the lung with a probang or we could have increased the oxygen pressure and ballooned the lung, but it was not necessary, and thus we had good room to work in with the lung almost collapsed. With the spinal anesthesia the patient was breathing very quietly. If there had been any difficulty I could have had more pressure by holding the patient's mouth closed or by increasing the pressure, or both. If the tumor had been adherent to the lung, and knowing it had a clear outline into the lung, I could very readily have divided the lung tissue between clamps and sewed it after the resection. I would have done just what Dr. Gilfillan did in his case where he had such a fine surgical result.

DR. C. NAUMANN MCCLOUD (St. Paul), retiring President, then read his Presidential Address entitled "Essential Hypertension," and asked for discussion from the members.

DR. MCCLOUD: Before presenting my paper, I wish to thank the Society for the honor shown me in electing me President of the Academy, where I have served during the past 15 months. It has been a very enjoyable year, and I particularly want to thank the members of the Executive Committee; they have been of very great help to me in conducting the proceedings of the organization, and in revising and putting in force the new Constitution. May I also state that our past Secretary, Dr. Carl Drake, has rendered a great deal of assistance to me, and I am sure that with the new efficient Secretary and the incoming officers, we should have a very profitable year before us.

I am very much interested in the subject of medical selection in connection with life insurance, and as the subject of hypertension has been causing us a great deal of perplexity, I thought this subject might be presented to the Academy tonight. In looking up the literature I was surprised to find how little there was published on the subject, and, though the President's address is not usually open for discussion, as there are several authorities on the subject of blood pressure here this evening, I would be very glad to have my paper discussed.

DISCUSSION

DR. H. T. NIPPERT (St. Paul): I would like to ask the Doctor about the normal diastolic pressure according to different ages; for instance, what is considered the normal diastolic pressure between 20 and 30 years and between 30 and 40, and so on.

DR. MCCLOUD: Speaking generally for life insurance companies, it would fluctuate, but it would vary 8 to 12 points above the average.

DR. E. L. GARDNER (Minneapolis): Dr. Bell's statistics on autopsied cases may place the percentage of cardiac deaths in hypertension rather high. In my experience, relatives object to postmortem examinations where the head is to be examined, and this may make the number of cerebral cases which are autopsied relatively much lower than those of cardiac failure.

DR. MCCLOUD: This is the experience of all medical directors, that they look upon the cause of death with a good deal of misgiving in a good many of these cases. For instance, just yesterday a man died a sud-

den death and the attending physician gave the cause of death as "edema of the lungs." From all symptoms it was coronary thrombosis, and the autopsy confirmed this diagnosis.

I would like very much to have Dr. Bell, for he can so beautifully do it, discuss the subject brought up by Dr. Gardner.

DR. E. T. BELL (Minneapolis): I am very much interested in Dr. McCloud's paper. The diastolic pressure has not been given as much attention as it deserves. I have not made much use of it in my studies because there seemed to be so much variation and irregularity that it could not be trusted in establishing the diagnosis of hypertension. If we use these two pressures we will find three groups: (1) A big group in which both the systolic and diastolic are elevated; (2) Cases in which the systolic is above normal and the diastolic is normal, and (3) A group in which the systolic is normal and the diastolic above normal. There are cases of hypertension with a normal diastolic pressure. The prognostic significance of the diastolic pressure must be determined from the experience of insurance companies. It will be a number of years before we have this information. Strange to say, there are not many statistical studies of very significant figures on the prognostic significance of high systolic pressure. Fischer found no increased mortality in accepted risks with systolic pressures of 140 to 750 mm. Hg.; but the death rate is much above the expected mortality with pressures above 150 mm. Hg. I have always considered a systolic pressure above 150, on repeated examinations, as hypertension.

Dr. Wetherby reported recently that in Dispensary patients over half above 50 years of age had a systolic pressure above 150 mm. Hg. Dr. Major found about the same proportion in the Ancker Hospital and Dispensary. Major found a systolic pressure above 150 mm. Hg. in about 40 per cent of the visitors above 50 years of age at the Ancker Hospital.

We are no longer sure what constitutes hypertension in the older age groups. Life insurance statistics deal largely with selected groups under 50 years of age. One of the first things we must find out is the normal pressure of the older groups. We have gone on the supposition that anything above 150 is hypertension. In about 12,000 postmortems which we have performed, 15 per cent of those over 50 years of age died from hypertension.

We have little or no knowledge as to the cause of primary hypertension. We have a lot of cases called secondary hypertension, practically all of which are secondary to kidney disease. That is the reason why Theodor Fahr is so strong on the kidney as the basis of hypertension. A fulminating primary hypertension is probably renal in origin; but in the great majority of hypertension cases there is not preliminary kidney disease but some unknown disturbance which keeps up a constant spasm of the arterioles of the body. Some 80 per cent of the arteriolar system is not involved. In the early cases of hypertension you can relax that spasm with nitrites. There is much evidence that the main thing is spasm, but what is the cause of that spasm? The only known factor in primary hypertension is inheritance. If it is inherited there must be some sort of physiological basis for it. Ninety per cent of the cases of hypertension appear after the age of 40 years. Hypertension is rare in young people.

DR. J. F. FULTON (St. Paul): Fundus changes constitute a very important factor in the clinical findings of hypertension so satisfactorily described by Dr. McCloud in his very valuable paper this evening. Asthenopia associated with come-and-go vision, together with highly congested disc and retina, is frequently an early symptom. High blood pressure and retinal arteriosclerosis frequently exist independently of each other. There are those who claim that fundus changes are met with in less than 50 per cent of the cases of hypertension; on the other hand, there are others who maintain that circulatory retinal changes are always found in persistent high hypertension if they are carefully investigated, commencing with spasm of the smaller vessels, resulting in narrowing of the arteries, kinking of the veins, engorgement and tortuosity, commencing twisting of the smaller arteries either between disc and maculæ or more frequently in the periphery, associated with the formation of small white spots in the retinal structure increasing in number as the condition lasts. Retinal hemorrhages demand careful study. The large flame-shaped hemorrhages taking place in the fibrous layers of the retina associated with edema are indicative of serious kidney pathology, while the small round punctate hemorrhages situated in the deeper retinal structures are met with and without hypertension. There is no relationship, however, between the height of blood pressure and the degree of arteriosclerosis. The white streak frequently seen along the course of the vessel is indicative of lymphatic complications.

I wish to call attention to the recently constructed self-illuminated ophthalmoscope as being of great value in detecting early changes in the arterioles in the retina.

DR. R. T. LAVAKE (Minneapolis): I would like to ask Dr. Bell about the early cases, those in young people. What does he mean when he says that he doesn't believe there is very much hypertension in the very young? How young? I have had several cases sent to me by men thinking that the patient had a toxemia of pregnancy, and I thought so too until I had followed them carefully for a considerable time. One was a girl of 20. She had a blood pressure of 160. On her father's side they all had hypertension. She never showed any kidney trouble so far as I could find, and she showed no nitrogen retention. She went all through pregnancy without any change in blood pressure.

DR. BELL: I had reference particularly to the college age. Hypertension is rare in college students. We had one case that began at 5 years of age; but I do not believe that it often begins in early life.

DR. LAVAKE: Another interesting feature was that this patient moved away and came back ten years later, and went through another pregnancy. The pressure had not varied in ten years.

DR. MCCLOUD (in closing): I would like to rise to the defense of the insurance statistics and our "be-

loved" examiners. In the 707,000 cases that were studied, those cases were not examined by "ordinary practitioners," but by experienced men, many of whom were medical referees and medical directors who spend nearly all of their time doing nothing but that sort of thing. However, that number constituted just a small portion of the number of cases examined; these were merely selected cases by that group of examiners. There were only 7,000 in that group that were ratable cases.

I wish to thank the members very much for their discussion of this paper.

The meeting adjourned.

R. T. LaVake, M.D.,
Secretary.

PROGRESS

Abstracts to be submitted to Section Supervisors.

Members are urged to abstract valuable articles which they run across in their reading and send the abstracts to the physicians in charge of the respective sections. In order to avoid duplication it would be well to communicate with one of the section supervisors before the article is abstracted.

MEDICINE

SUPERVISORS:

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THOMAS A. PEPPARD,
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THE PHYSIOLOGICAL MECHANISM OF PNEUMOTHORAX: F. Dumarest (Revue de La Tuberculose, 1929, X177). According to Forlanini the primary cause of the progressive nature of ulcerative pulmonary tuberculosis is the respiratory traumatism which is exerted upon a tissue that has lost its capacity to expand. Dumarest's former assistant, Parodi, has recently published a book on Pneumothorax which is concerned with the relations of the respiratory mechanism to the technic of the procedure.

As regards normal physiology, Parodi considers the movements of the thorax dependent on the elasticity of the lung: first, the structural elasticity of retraction, due to elastic fibres in the lung, which is effective during expiration; and, secondly, the functional elasticity which distends the lung during inspiration, brought about by the enlargement of the thorax and which represents the capacity of the lung for change in form.

It is easier to immobilize the apex of the lung than the base, and infiltrated rather than healthy areas because their capacity for retraction is increased while their power of distention is lessened. The various parts of the lung are independent of each other and this makes selective collapse possible. It is difficult to collapse healthy lung tissue. In such tissue there may even be reversed pressure due to interference with expiration by closed bronchioles and by muscular bands. In order that healthy lung tissue be collapsed, the air in the alveoli must be absorbed or expelled. Gradual relaxation of the muscle fibres increases the degree of possible collapse.

In the child, collapse can be brought about more easily as the elasticity of retraction is much greater than that of distention.

The diaphragm is an index of the elasticity of the lung structure. When there is infiltration, the diaphragm ascends; when the elasticity of the lung is diminished, as in emphysema, the diaphragm descends

The rôle of the mediastinum is no less important In the dog the mediastinum is very movable and a complete collapse of the lung cannot be secured without causing serious cardiac and vascular difficulties.

In the tuberculous patient the mediastinum is usually more fixed than in a healthy person, and an immediate complete collapse becomes possible. The degree of mediastinal fixation is therefore always important, but less so in bilateral pneumothorax.

When a pneumothorax is started, the amount and the rapidity of penetration of the gas under atmospheric pressure gives valuable information as to the state of retraction and the capacity for distention of the lung.

The rapidity with which the gas is taken and the amount of collapse secured are dependent upon the degree and nature of the lesions and of the lung tissue, whether more or less emphysematous. Neutralization of the tendency to expand is dependent on the state of the muscles and the power to inspire.

To produce a selective collapse it is necessary to introduce only that amount of gas which will neutralize the structural elasticity in the diseased area without seeking to reduce the functional elasticity of expansion in the healthy part of the lung. This can be brought about only when negative pressure is used in giving the pneumothorax..

Parodi has devised an apparatus called an "ergomanometer" which permits a closer analysis of the factors concerned in collapse than does the ordinary manometer.

A. T. Laird, M.D.

INDICATIONS FOR THORACOPLASTY: Karl Fischell (Amer. Rev. of Tuber., 1928, XVIII, 552/4). The more extensive the lesion on the compressed side, the smaller is the amount of normal lung tissue that will be affected by the operation and the better will the functional deficit be compensated by the increased activity of the better side.

Acute cases with short duration are not suitable for rib resection before a tendency to retraction is demonstrated. Operation is contraindicated in the stage of defense and should be attempted when the stage of re-

pair is reached when the heart and lung have had time to adapt themselves to diminution of the breathing space and are ready for increased compensatory function and, above all, retraction in different parts of the chest, thickening of the pleura, displacement and fixation of the thoracic viscera have developed, which indicate the necessary tendency to fibrosis which can be intensified by thoracoplasty.

Thoracoplasty acts on the chest wall, phrenicotomy immobilizes the floor. We know of no means to make the mediastinum resistant unless it is fixed by adhesions. If it is not fixed, paradoxical breathing or mediastinal flutter occurs.

Fever, cough, dyspnea and rapid pulse and palpitation are usually observed during the first week, the combined effect of shock, pulmonary and cardiac embarrassment and increased absorption of toxins.

The cardio-pulmonary reaction will be milder if the compression is done in two or three stages.

Other contributory factors to the postoperative reaction are sputum retention, aspiration pneumonia, and toxic crises. The latter resembles a true tuberculin reaction and will be mild or absent in chronic cases.

The risks of operation are greater if performed at too early a period. The case must be mature, as a cataract must be before it is removed.

In exudative lesions compression is not advisable or possible.

Marked constitutional symptoms such as high fever, dyspnea, cyanosis, and rapid pulse represent a formal contraindication.

The general condition of the patient must be taken into account. He must have a well functioning heart, and meet the requirements of a good surgical risk as to blood pressure, hemoglobin, red and white count and coagulation time. Contraindications on account of the better side must be stricter than in pneumothorax. It is wise to test the adaptability of the other side by phrenicotomy first.

Classical contraindications are extensive tuberculosis in other organs, extreme youth or age and cardiac and renal disease and diabetes. The author considers involvement of the larynx or of the intestines a contraindication. Myocarditis and valvular lesion of the heart and contraindications.

A. T. Laird, M.D.

INFRACLAVICULAR FOCI OF INFILTRATION AS EVIDENCE OF INCIPIENT TUBERCULOSIS OF THE LUNGS IN YOUNG ADULTS: Prof. Dr. H. Assmann (Radiology, XIV, Feb. 1930, 93). The author describes an isolated, rounded focus of varying density, usually situated just below the clavicle, in the lateral part of the pulmonic field which he considers to be a characteristic type of pulmonary lesion in the early stage of tuberculosis, particularly in young adults. He first described this lesion in 1922 and again in 1924. The apices are uninvolved in this type of tuberculosis. The position of this focus at a distance below the surface presents few or no physical signs, and the general clinical signs are also often minimal.

The anatomic basis is assumed to be the beginning tuberculous pneumonic infiltration, *i.e.*, an exudative process which often leads to caseation and central necrosis and thus to cavity formation which is well demonstrated on the *x*-ray plate. The author does not consider this a primary focus of infection since that usually occurs in childhood and is generally overcome, but rather a re-infection in an aërogenic manner.

The prognosis is generally favorable under proper treatment and the shadow of the focus may disappear, leaving only a very small linear scar.

Jacob Sagel, M.D.

ROENTGENOLOGY

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RADIOLOGY OF THE THORAX: Samuel Brown, M.D. (Rad., Dec., 1929, XIII, 515). The author feels that antero-posterior and lateral views of the chest are superior to stereoscopic plates except in early tuberculosis. There is a constant relationship between the size of the heart and the thorax and this is expressed in a formula developed by the author. The *x*-ray anatomy of the thorax is well described. A knowledge of the exact position and relationship of the intrathoracic structures in the antero-posterior and lateral views is of great value in recognizing abnormal changes affecting any of the structures. The points discussed are well illustrated by twenty-four plates taken in the antero-posterior and lateral views.

Jacob Sagel, M.D.

THE ROENTGENOLOGIC MANIFESTATIONS OF INTRACRANIAL DISEASE: John D. Camp, M.D. (Rad., Dec., 1929, XIII, 484). In this type of work, stereoscopic plates in the lateral view, antero-posterior, and postero-anterior plates should be taken. Frequently, additional views of special structures are necessary. In order of their importance the various roentgenologic signs of intracranial diseases may be grouped as follows: (1) calcification within the lesion itself, (2) localized changes in the bone such as destruction, proliferation, or both, (3) changes in the sella turcica, (4) changes in the bone resulting from increased intracranial pressure, (5) displacement of the pineal shadow, (6) air in or about the lesion, (7) increased vascularity of bone.

Calcification within the lesion occurs in only a small percentage of cases. The usual change in the

bones overlying a contiguous tumor is a localized thinning due to pressure. The lesions producing deformities of the sella turcica are: (1) intrasellar tumors involving the pituitary gland, (2) suprasellar lesions, (3) disease of the sphenoid bone. Displacement of the shadow of the pineal gland is a very important sign in diagnosis. Increased vascularity of bone must be interpreted cautiously due to marked variations in the prominence of the diploë and other vascular markings. This sign by itself is not worth much. The article is well illustrated and a large bibliography is appended

JACOB SAGEL, M.D.

PEDIATRICS

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EARLY RHEUMATIC INFECTIONS IN CHILDHOOD: C. C. McLean, M.D., Birmingham, Ala. (Arch. of Ped., November, Vol. XLVI, No. 11). Early rheumatic infection of childhood is characterized by an insidious onset. Many children complain of slight pains in the legs, feet or joints. They begin to drop objects and have difficulty in writing. Examination will show an anemic child who is nervous, excitable, high strung and underweight. Often no murmur is heard, but the heart action is rapid, with the apex impulse exaggerated.

When a diagnosis of rheumatic infection is made it is not always given the importance that it deserves. Feer states that 80 or 90 per cent of all cases of rheumatic infection will have a certain degree of heart involvement.

The most common symptoms are: (1) Poor appetite and failure to gain weight; (2) nervousness, irritability and fatigue; (3) repeated attacks of tonsillitis and nasopharyngeal infection with temperature; (4) pain in legs, joints, or stiffness of limbs. Seventy-one per cent have soft blowing systolic murmurs, the second heart sound being accentuated in each case. There were 103, or 87.3 per cent, underweight for their height.

Every child with manifestations of an early rheumatic infection should be given the benefit of the doubt and managed accordingly. The foci of infection should be found and removed when possible. Rest in bed and forced feeding is very important in this type of case. They are given a six-meal diet, with special emphasis on liver, spinach, prunes, whole wheat breads and cereals. They are also given iron and cod liver oil.

Every child with manifestations of an early rheumatic infection is a potential cardiac patient, and should be managed accordingly.

R. N. ANDREWS, M.D.

BOOK REVIEWS

Books listed here become the property of the Ramsey and Hennepin County Medical libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

BOOKS RECEIVED FOR REVIEW

GENERAL MEDICINE. Edited by George H. Weaver, M.D., et al. 829 pages. Illus. Cloth, $3.00. Chicago: The Year Book Publishers, 1929.

COMMENT RAJEUNIR. Dr. H. Jaworski. Albin Michel, Editor. 250 pages. Paper, 12 francs. Paris: Albin Michel, 22, Rue Huyghens, 1930.

GETTING WELL AND STAYING WELL. Dr. John Potts. 221 pages. Cloth, $2.00. St. Louis: The C. V. Mosby Company.

SYMPTOMS OF VISCERAL DISEASE. Dr. Francis Marion Pottenger. 426 pages. Illus. Cloth, $7.50. St. Louis: The C. V. Mosby Company.

OUTLINE OF PREVENTIVE MEDICINE. Prepared under the auspices of the Committee on Public Health Relations, New York Academy of Medicine. Editorial committee: Frederic E. Sondern, Charles Gordon Heyd and E. H. L. Corwin. 398 pages. Price, $5.00. New York: Paul B. Hoeber, 1929.

This is an interesting and instructive book. Preventive medicine is a field that each practitioner should know and practice, and this book covers every field of medicine. There are twenty-one chapters, each chapter by a different author. The ground is well covered in a brief and interesting way and offers to the specialist an opportunity of covering the newest in other fields in a short period of time. It is a book worth reading.

A. N. BESSESEN, JR., M.D.

TULAREMIA. Walter M. Simpson, M.S., M.D., F.A.C.P. 162 pages. Illus. Cloth, $5.00. New York: Paul B. Hoeber, 1929.

This book is an extremely interesting presentation of a disease discovered and developed entirely by our own men. The printing is large and easy to read, 136 pages. It is fully illustrated and a large bibliography gives credit to each worker in the field. This book is not only interesting but instructive, and should be read by every American physician because of its inspiration. It promises a delightful evening at home and one the wife will enjoy as well, for it reads like a novel.

ALFRED N. BESSESEN, JR., M.D.

THE TREATMENT OF FRACTURES. Lorenz Böhler, Vienna. 185 pages, 234 illustrations. Cloth, $5.00. Authorized English translation by M. E. Steinberg.

Dr. Böhler has written an excellent book, covering the entire field of fractures. After carefully reading this book, it is the opinion of the reader that Dr. Böhler has given us many new ideas, which may somewhat revolutionize our treatment of fractures. It is a pronounced departure from the usual orthodox fracture treatment. While the author stresses non-operative treatment, his methods really are more radical than our present operative treatment. Dr. Bohler has had the fortunate experience of treating many hundreds of fractures in Austria, under conditions which are not available in this country. Undoubtedly his methods are excellent in the hands of well trained men, with good hospital facilities, and a surgical team of six or eight people to carry out his method of treatment.

He emphasizes the use of local anesthesia, and interfragmental anesthesia almost universally. He favors the non-padded cast, recommends large amount of equipment, and goes into minute detail as to all the requisites necessary for treatment. He emphasizes immediate, forcible reduction, and uses skeletal traction in most all of his fractures. He introduces several new splints, both for bed treatment and ambulatory treatment. He has outlined his principles very clearly, but many of his methods are peculiar to his clinic alone. He has emphasized function very thoroughly, and this alone is a great contribution. Some of his operative methods, especially those for the treatment of pseudoarthrosis, are obsolete.

I believe this book is a definite contribution to the treatment of fractures, and I would recommend it to all men who have had training and experience in fracture work, but it is certainly a book to be kept out of the hands of people who rarely treat fractures, and who might become enthusiastic about these methods.

E. A. REGNIER, M.D.

PHYSICIAN WANTED—Young physician to do general practice and resident work in clinic operating sixty-bed hospital in South Dakota. Excellent opportunity for clinical experience and good permanent clinic affiliation. Moderate salary offered for first year with opportunity for permanent connection following. Address D-58, care MINNESOTA MEDICINE.

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